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LONGITUDINAL SECTION OF PYLORUS FROM CASE OF CONGENITAL HYPERTROPHIC STENOSIS OF PYLORUS.

M mucous membrane; SM submucous tissue; CM circular muscular coat; LM longitudinal muscular coat; I intestinal wall; 8 diam.

TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

SIXTEENTH SESSION

HELD AT DETROIT, MICH., MAY 30, 31 AND JUNE 1, 1904

EDITED BY

LINNÆUS EDFORD LA FÉTRA, M.D.

VOLUME XVI.

REPRINTED FROM
ARCHIVES OF PEDIATRICS
1904–1905

BOSTON MEDICAL
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PRESIDENTS.

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. Wm. P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. Wm. D. Booker, M.D.
1902. W. S. Christopher, M.D.
1903. J. P. Crozer Griffith, M.D.
1904. Augustus Caillé, M.D.
1905. C. G. Jennings, M. D.

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F. Forchheimer, M.D.
J. C. Wilson, M.D.
F. M. Crandall, M.D.
L. Emmett Holt, M.D.
F. S. Churchill, M.D.

MEETING PLACES.

1888. Washington, D. C. (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 and 25.
1892. Boston, May 2, 3 and 4.
1893. West Point, N. Y., May 24, 25 and 26.
1894. Washington, May 29 and June 1.
1895. Virginia Hot Springs, May 27, 28 and 29.
1896 Montreal, May 25, 26 and 27.
1897. Washington, May 4, 5 and 6.
1898. Cincinnati, June 1, 2 and 3.
1899. Deer Park, June 27, 28 and 29.
1900. Washington, May 1, 2 and 3.
1901. Niagara Falls, May 27, 28 and 29.
1902. Boston, May 26, 27 and 28.
1903. Washington, May 12, 13 and 14.
1904. Detroit, May 30, 31 and June 1.
1905. Lake George, N. Y., June 19, 20 and 21.

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Secretary.....SAMUEL S. ADAMS, M.D.
Treasurer.....J. PARK WEST, M.D.
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JOHN LOVETT MORSE, M.D.

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ACKER, GEORGE N., M.D.....913 Sixteenth Street, Washington
ADAMS, SAMUEL S., M.D.....1 Dupont Circle, Washington
BAINES, ALLEN, M.D.....194 Simcoe Street, Toronto
BLACKADER, A. D., M.D.....236 Mountain Street, Montreal
BOOKER, WILLIAM D., M.D.....208 West Monument Street, Baltimore
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DORNING, JOHN, M.D.....111 West Forty-eighth Street, New York
EATON, PERCIVAL J., M.D.....131 N. Highland Avenue, E. E., Pittsburgh
EDSALL, D. L., M.D.....346 South Sixteenth Street, Philadelphia
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GRAHAM, E. E., M.D.....1713 Spruce Street, Philadelphia
GRIFFITH, J. P. CROZER, M.D.....1810 Spruce Street, Philadelphia
HAMILL, S. McC., M.D.....1822 Spruce Street, Philadelphia
HAND, ALFRED, JR., M.D.....1724 Pine Street, Philadelphia
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HUBER, F., M.D.....209 East Seventeenth Street, New York

JACOBI, A., M.D.....19 East Forty-seventh Street, New York
JENNINGS, CHARLES G , M.D.....457 Jefferson Avenue, Detroit
KERLEY, CHARLES G., M.D.....113 West Eighty-third Street, New York
KOPLIK, HENRY, M.D.....692 Madison Avenue, New York
LADD, MAYNARD, M.D.....220 Marlborough Street, Boston
LA FÉTRA, LINNÆUS E., M.D.....58 West Fifty-eighth Street, New York
MCCOLLOM, JOHN H., M.D.....745 Massachusetts Avenue, Boston
MARTIN, C. F., M.D.....33 Durocher Street, Montreal
MILLER, D. J. MILTON, M.D.....1801 Pine Street, Philadelphia
MORRILL, F. GORDON, M.D.....181 Beacon Street, Boston
MORSE, J. LOVETT, M.D.....70 Bay State Road, Boston
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OSLER, WILLIAM, M.D.....1 West Franklin Street, Baltimore
PUTNAM, CHARLES P., M.D.....63 Marlborough Street, Boston
RACHFORD, B. K., M.D.....323 Broadway, Cincinnati
ROTCH, THOMAS MORGAN, M.D.....197 Commonwealth Avenue, Boston
SAUNDERS, E. W., M D.....1635 South Grand Avenue, St. Louis, Mo.
SEIBERT, A., M.D.....114 East Fifty-seventh Street, New York
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SNOW, IRVING M., M.D.....476 Franklin Street, Buffalo
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TOWNSEND, CHARLES W., M.D.....76 Marlborough Street, Boston
WENTWORTH, A. H., M.D.....416 Marlborough Street, Boston
WEST, J. PARK, M.D.....3608 Belmont Street, Bellaire, Ohio
WESTCOTT, THOMPSON S., M.D.....1833 Spruce Street, Philadelphia
WILLIAMS, HAROLD, M.D.....528 Beacon Street, Boston
WILSON, W. REYNOLDS, M.D.....1709 Spruce Street, Philadelphia
WINTERS, J. E., M.D.....25 West Thirty-seventh Street, New York
YALE, LEROY M., M.D.....432 Madison Avenue, New York

HONORARY MEMBERS.

DR. JOHN THOMPSON.....Edinburgh, Scotland
DR. GEORGE F. STILL.....London, England
DR. HENRY ASHBY.....Manchester, England
DR. O. HEUBNER.....Berlin, Germany
DR. THEODOR ESCHERICH.....Vienna, Austria

Deceased.

JOHN A. JEFFRIES, M.D.
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.
Born, April 20, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.
Born, October 15, 1827,
Died, June 9, 1897.

JOSEPH O'DWYER, M.D.
Born, October 12, 1841,
Died, January 7, 1898.

JOHN HENRY FRUITNIGHT, M.D.
Born, November 9, 1851,
Died, December 18, 1900.

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MINUTES OF THE SIXTEENTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY.

Held at Detroit, Mich., on May 30, 31 *and June* 1, 1904.

The following members were present:—I. A. Abt, M.D., Chicago; G. N. Acker, M.D., Washington; S. S. Adams, M.D., Washington; A. Baines, M.D., Toronto; A. Caillé, M.D., New York; F. S. Churchill, M.D., Chicago; A. C. Cotton, M.D., Chicago; F. M. Crandall, M.D., New York; J. Dorning, M.D., New York; F. Forchheimer, M.D., Cincinnati; R. G. Freeman, M.D., New York; L. E. Holt, M.D., New York; A. Jacobi, M.D., New York; C. G. Jennings, M.D., Detroit; C. G. Kerley, M.D., New York; S. M. Ladd, M.D., Boston; L. E. La Fétra, M.D., New York; J. L. Morse, M.D., Boston; W. P. Northrup, M.D., New York; C. P. Putnam, M.D., Boston; B. K. Rachford, M.D., Cincinnati; T. M. Rotch, M.D., Boston; I. M. Snow, M.D., Buffalo; J. P. West, M.D., Bellaire; J. E. Winters, M.D., New York.

FIRST SESSION, MAY 30TH, 10 A.M., RUSSELL HOUSE.

Dr. Irving M. Snow, of Buffalo, read a paper on "Intussusception in a Baby of Seven Months: Elimination of the Invaginated Bowel by Gangrene; Cure."

Discussion by Drs. Putnam and Caillé.

Dr. S. Maynard Ladd, of Boston, presented a paper entitled, "An Investigation of the Influence of Laboratory Feeding on 216 Infants with Diseases of the Gastroenteric Tract," with special reference to the Weight Index.

Discussion by Drs. Kerley, Freeman, Crandall, La Fétra, Putnam, Holt and Ladd.

Dr. L. Emmett Holt, of New York, read a paper entitled, "Disturbances from High Fat in Infant Feeding."

Discussion by Drs. Northrup, Kerley, Crandall, Morse, Rotch, Rachford, La Fétra, Putnam, Winters, Jacobi and Holt.

Dr. John Dorning, of New York, presented a paper on "Congenital Hypertrophic Stenosis of the Pylorus."

Discussion by Drs. Northrup, West, Morse, Holt, Rotch, Caillé and Dorning.

SECOND SESSION, MAY 30TH, 2 P.M.

Dr. Augustus Caillé, of New York, read a paper entitled, "A Cure of Chronic Nephritis following Renal Decapsulation: Second Report."

Discussion by Drs. Kerley, Putnam, La Fétra, Forchheimer and Caillé.

Dr. A. C. Cotton, of Chicago, read a paper entitled, "Some Observations on Epidemic Vulvovaginitis in Infants and Children."

Discussion by Drs. Northrup, Kerley, Jacobi, La Fétra, Acker, Abt, Caillé, Adams and Cotton.

Dr. Samuel S. Adams, of Washington, presented a paper entitled, "A Case of Carbolic Acid Poisoning Simulating Acute Scarlatinal Nephritis."

Dr. B. K. Rachford read a paper on "Gastrointestinal Toxemia."

Discussion by Drs. Jacobi, Forchheimer, Northrup and Rachford.

Dr. Charles G. Kerley presented a paper entitled, "Clinical Manifestations of Acute Otitis in Children: A Study of Fifty-one Cases in Private Practice."

Discussion by Drs. Rotch, Cotton, Morse, Abt and Kerley.

7 P.M., DINNER AND ENTERTAINMENT BY THE WAYNE COUNTY MEDICAL SOCIETY. JOINT MEETING.

THIRD SESSION, MAY 31ST, 9:30 A.M., RUSSELL HOUSE.

Dr. William P. Northrup, of New York, read a paper on "Nervous Exhaustion in Infants."

Discussion by Drs. Cotton, Caillé, Putnam, Adams, Freeman, Churchill and Northrup.

The annual address of the president, entitled, "The Influence of American Pediatric Societies in Promoting the Welfare of American Children," was read by Dr. Augustus Caillé, of New York.

Dr. J. Lovett Morse, of Boston, presented a paper entitled, "Analysis of One Hundred and Eighteen Cases of Lobar Pneumonia in Infancy."

Discussion by Drs. Kerley, Abt, Churchill, Holt, Adams, Northrup and Morse.

Dr. Rowland G. Freeman, of New York, read a paper on "Acute Pyelitis in Infancy."

Discussion by Drs. Morse, Abt, Rotch, Dorning, Crandall, Holt, Jacobi and Freeman.

Dr. F. Forchheimer, of Cincinnati, presented a paper entitled, "Acute Myocardial Insufficiency in Some Infections of Children."

Discussion by Drs. Rotch, Jacobi and Forchheimer.

FIFTH SESSION, JUNE IST, IO A.M., COUNTRY CLUB.

Dr. Linnæus E. La Fétra, of New York, read a paper on "Bronchial Asthma in Infants and Children: a Study of Forty-three Cases."

Discussion by Drs. Jennings, Holt, Crandall, Caillé and Northrup.

Dr. William P. Northrup, of New York, presented a paper entitled, "Dyspnea, Central, Peripheral, Obstructive: Cases."

Discussion by Drs. Holt, Crandall, Abt, Caillé and Northrup.

Dr. F. S. Churchill, of Chicago, read a paper entitled, "Acute Leukemia, with Report of a Case."

Dr. L. Emmett Holt, of New York, read a paper entitled, "Adenosarcoma of the Liver."

Discussion by Drs. Abt, Northrup and Caillé.

Dr. Samuel S. Adams presented, for Dr. H. B. Sheffield, the "Radiograph of a Case of Abdominothoracopagus."

The following papers were read by title:

Dr. A. H. Wentworth, of Boston, "A Study of the Metabolism of Atrophic Infants and Children."

Dr. H. L. K. Shaw, of Albany, "A Case of Congenital Pyloric Stenosis in an Infant: Operation: Recovery."

Dr. Henry D. Chapin, of New York, "The Management of Convalescent Infants and Children, after Leaving the Hospital."

Dr. Henry Koplik, of New York (*a*) "The Rachitic Hand"; (*b*) "The Clinical Features and Diagnosis of Tuberculous Meningitis"; (*c*) "Percussion of the Head in Infants and Children, with Special Reference to the Value of the Macewen Sign in Meningitis."

Dr. E. W. Saunders, of St. Louis, "A Plea for the More Time-

ly Use of Intubation in Laryngeal Stenosis, and for the Use of Tracheotomy in Certain Neglected Cases."

Dr. D. J. M. Miller, of Philadelphia, "Hypertrophic Osteoarthropathy in Children; its Relationship with the Ordinary Clubbed Fingers of Chronic Heart and Lung Affections."

Dr. Francis Huber, of New York, "Observations on Cerebrospinal Meningitis."

Dr. T. M. Rotch, of Boston, "Some Considerations Regarding the Problems of Infant Feeding."

FOURTH (EXECUTIVE) SESSION, MAY 31ST, 2 P.M., DETROIT CLUB.

Dr. C. P. Putnam, Chairman of the Council, presented the following report of the Council:

It is recommended that the resignations of the following gentlemen be accepted with regret:—Dr. J. C. Wilson, of Philadelphia; Dr. Henry Jackson, of Boston; Dr. Alfred Stengel, of Philadelphia. The recommendation was adopted.

The following officers were elected for the ensuing year:—President, Dr. C. G. Jennings; First Vice-President, Dr. C. G. Kerley; Second Vice-President, Dr. T. S. Westcott; Secretary, Dr. Samuel S. Adams; Treasurer, Dr. J. Park West; Recorder and Editor, Dr. L. E. La Fétra.

Dr. J. P. C. Griffith was appointed a member of the Council to succeed Dr. J. C. Wilson, resigned. Dr. J. L. Morse was elected a member of the Council to succeed Dr. C. P. Putnam, retired.

It was recommended that the next annual meeting be held at Sagamore Inn, Lake George, N. Y., June 19, 20 and 21, 1905.

Dr. Percival J. Eaton, of Pittsburg, Pa., was elected to membership.

Dr. L. E. Holt moved that the secretary be instructed to furnish the mailing list to all the members of the society, that they might recommend additions to the list, particularly to dispose of the surplus volumes in the hands of the secretary. Adopted.

Dr. Putnam moved that the Council be authorized to furnish 200 additional volumes of the Transactions, if it was thought necessary. Adopted.

It was recommended that E. B. Treat & Co. be given the work of publishing the transactions of the Society as it had been done so satisfactorily this year.

The secretary was instructed to express the thanks of the

Society to the Detroit, Yondotega and Country Clubs of Detroit, and to Parke, Davis and Co. for courtesies extended.

Upon motion the retiring president was thanked for the efficient and impartial manner in which he had conducted the affairs of the Society.

Upon motion of Dr. Jennings, the thanks of the Society were expressed to the secretary for the earnest efforts to make this meeting a success.

Upon motion, Dr. Jennings was thanked for his efforts to make the meeting successful and pleasant to the members.

Meeting adjourned.

LINNÆUS E. LA FÉTRA, M.D.,
Recorder.

THE INFLUENCE OF AMERICAN PEDIATRIC SOCIETIES IN PROMOTING THE WELFARE OF AMERICAN CHILDREN.

PRESIDENTIAL ADDRESS.

BY AUGUSTUS CAILLÉ, M. D.,
New York.

GENTLEMEN: First, let me express my gratitude for your kindness in conferring upon me the honor of the presidency of the American Pediatric Society.

According to Article I. of the constitution and by-laws, the American Pediatric Society has for its object the advancement of the physiology, pathology and therapeutics of infancy and childhood.

On various occasions my predecessors in office have interestingly reviewed the past work of the Society, and have pointed out to what extent our labors have been recognized by the profession at large.

It seems to me that after fifteen years of prosperous existence the time has arrived when the weight and authority of our Society, in addition to being felt by our professional colleagues, should be made to extend to the millions of homes throughout the land. In other words, the American Pediatric Society should now be ready to assume broader obligations and to grapple with larger problems. To shape and promote the public understanding regarding sanitation of the child's environment, and to supervise from a medical standpoint the education of American children should be one of the chief aims of the American Pediatric Society.

The life of the child runs its course in the home, the school, the playground, and, unfortunately, in the workshop; and the myriad of problems that confront us as guardians of the child may, after all, be condensed into one grand effort, namely, that of sanitation. I, therefore, beg to be permitted to point out, briefly, some of the more important matters to which we as a body may direct our attention.

THE OVERHEATING OF AMERICAN HOMES AND THE FEAR OF CATCHING COLD.

The overheating of living rooms and sleeping rooms in homes and temporary homes, such as boarding schools, is one of the most fruitful causes of ill-health in children. The fear of breathing fresh air still holds possession of the multitude, as it did in former times, when microbial infection was a terra incognita; and the comparative few who have dared to advocate the breathing of cool, fresh air are looked upon as little short of dangerous advisers by the wiseacres of the domestic hearth.

A "cold," although an every-day occurrence, has, like the Monroe Doctrine or the Immortality of the Soul, not as yet received a satisfactory explanation. Areas of minor resistance or irregularities of capillary circulation are inherent or brought on in various individuals. If cold feet produce catarrh of the upper air passages, or if a hair-cut is followed by a stiff neck or sore throat, such an irritation is probably reflected or transmitted to an area of minor resistance, and the resulting local hyperemia favors infection or the invasion of such microbes as happen to be present, though latent. In this manner we probably contract colds of the upper air passages, which, according to localization, are called rhinitis, pharyngitis, laryngitis and bronchitis. *The susceptibility to cold-catching can be markedly reduced by a judicious hardening process in connection with proper alimentation.* To use a cold bath or a cold douche for hardening nurslings is probably deleterious; but in older children a hardening process by cold douching is undoubtedly beneficial, and will strengthen the child's forces and permit it to overcome inclement influences of various kinds. The susceptibility to cold-catching cannot be reduced, however, in homes in which overheating of the living apartments is tolerated. Under such unfavorable conditions, infants and older children are seldom free from catarrhal conditions of the respiratory organs, and many of the socalled difficult feeding cases, acute and subacute indigestion, resist management and drag on from bad to worse for the same reason, and improve as soon as the children are taken out of doors. In this connection it may be well to state that one of the most dangerous methods of heating a room is the portable gas stove not connected with a flue. The products of combustion remain in the room and vitiate the air to such an extent that adults and older children suffer from general malaise, and infants suffer seriously from various forms of ill-defined sickness.

Overheating and faulty heating of living apartments should be condemned by the medical profession at every opportunity which may present itself, and fresh-air treatment of healthy and sick children should be insisted upon. In this respect Boston is setting a good example of late, inasmuch as some of the wealthy and aristocratic families of the Back Bay district are putting their babies to sleep out of doors, on roofs, balconies and wide window-sills. The method is begun when the baby is about two months old, and is continued until the child is five or six years old. The infant is wrapped up like a pappoose and is laid on a hair mattress in a box or basket, and so strapped that the arms and legs have full freedom. The child is warmly covered with blankets and above the bed is an awning shielding it from wind and direct sun rays. It is stated that during the last winter, notwithstanding the severity of the weather, there were but few days when the babies were not placed out of doors.

DISINFECTION AND THE SANITARY CODE.

In case of contagious disease, physicians are required to make a report; and on the report of a case, a health inspector endeavors to trace the origin of the disease, provides disinfectants, and reports the case at school. The disinfection of living apartments, with filthy carpets and hangings, cracked and papered walls, soiled upholstered furniture, and the thousand and one odds and ends, is an illusion, as practised in our large cities. The removal of a convalescent child to another room or to a neighboring apartment during the fumigation process, which takes ten to twelve hours, theoretically and practically nullifies all efforts to destroy sources of infection or to stamp out contagious disease. The compulsory cleansing of an apartment with soap and water, as practised in some of the larger cities of Europe, has more real value than fumigation with sulphur. One of the principal means of the spread of contagious disease is overlooked and may be found in the cloakrooms of factories and schools, in which the wraps and outer garments, often wet and damp, of the adults and children are heaped together. The same may be said of books in schools and libraries. They are frequently soiled with the nasal discharge of the sick, and as yet a practical and thorough method of disinfection has not been elaborated.

The practice of compelling parents to keep children convalescent from diphtheria indoors and confined to the sickroom, until all bacilli have disappeared from the throat, is in my opinion

unreasonable, because it deprives the convalescents of the tonic effects of fresh air which they require, and because the infectious nature of diphtheria bacilli found during convalescence and later on *has not been proven.*

Professor Dunbar, the Director of the Hamburg Hygienic Institute, recently informed me that school children are permitted to re-enter the schools in Hamburg very soon after the clinical symptoms of a diphtheritic infection had subsided, and that no notice is taken of the presence or absence of diphtheria bacilli in such cases, because careful investigation of such factors in schools and asylums had shown that the so-called bacillary precautions have not been followed by noteworthy results.

One of the most cruel results of the Sanitary Code as practised in some of our large cities is the taking away of children suffering from ordinary every-day eruptive fevers and transferring them to a suburban department hospital, against the will of the parents, when such cases are reported from a hospital or from an apartment, some portion of which is utilized for business purposes.

This enactment is directed chiefly against the poor, and has given rise to so much bitterness of feeling that in the very many instances the fear of the compulsory removal of a child to a department hospital has prevented parents from calling in a physician and has kept well-meaning and humane physicians from reporting such cases.

It must be conceded that in many instances it would be to the interest of all concerned, if cases of communicable disease could be treated in a proper hospital; but to compel parents to give up their children in ordinary cases of illness is wrong. It is far better to take no stringent precaution than to cast discredit upon sanitation in general, by enforcing cruel and unnecessary laws. If the community demands isolation under all circumstances, the authorities should insist upon securing proper accommodations for mother and child. A free discussion and criticism of the Sanitary Code as interpreted by some of our Health Boards would appear opportune.

SCHOOL HYGIENE AND SCHOOL INSPECTION.

Medical school inspection has been systematically carried out in France and Germany for half a century. It is a public question and merits the constant attention of medical and especially pediatric societies. Daily medical inspection of the schools of the

city of New York received its first practical agitation in 1890, at a public meeting of the Pediatric Section of the New York Academy of Medicine, under the Chairmanship of the writer. It was successfully inaugurated in Boston in 1894, in New York in 1897, in Philadelphia in 1898, and, subsequently, in many other large centres throughout our country. The inspection is made during the early school hours, and children found to be ill are sent home with a card informing the parents that their child needs medical treatment.

School inspection had primarily for its object the early detection of communicable disease, but in its logical development it now embraces, or should embrace, in its broadest sense, the hygienic guardianship of school children. It is of far-reaching importance as regards the welfare of the masses from a hygienic and economic standpoint, because it is a powerful factor in educating children and parents. In order fully to appreciate the far-reaching beneficial effects of school inspection, we need only to look over what has been accomplished within the few years that it has existed in our country.

A rational school inspection reveals unsanitary conditions in general, existing in schools, day nurseries and kindergartens; it is a powerful factor in promoting the cleanliness of the scholars, and insures proper attention to existing parasites, skin lesions and obnoxious habits. Early detection of measles, scarlet fever, chicken-pox, whooping-cough, diphtheria, tuberculosis, contagious ophthalmia and other communicable disease is one of the features of school inspection. Mouth-breathers are detected at a glance and children suffering from adenoids, enlarged tonsils or other nasal obstruction are thus put into the way of receiving the benefits of proper treatment.

Backward and neurotic children, anemic children, with simple loss of appetite, or those suffering from headache, chorea, neurasthenia, eye strain from visual defects, or partial deafness, are singled out readily.

There are certain diseases brought about by faulty positions in sitting, writing, study and standing. For instance, cramped position causes round shoulders, scoliosis, flat chest, myopia, etc. It is estimated that fully 90 per cent. of curvature, not due to bone disease, is developed during school life from this cause alone.

The early detection of vaginal discharges of gonorrheal origin in young children in day nurseries, kindergartens and primary

classes in public schools is of the utmost importance in preventing the spread of this malady and its complications, such as contagious ophthalmia, etc.

To illustrate the dangers of gonorrheal infection in children in public schools, the following authentic instance is cited: In a public institution in New York City harboring 850 children, 130 cases of socalled leucorrhea developed in two months, which proved to be gonorrheal in nature. A large number of these children attended public schools. After numerous complaints by parents of children subsequently infected had been made, the matter was carefully investigated and proper isolation was effected.

When medical inspection is combined with rational elementary instruction in hygiene, as inaugurated in the state of Michigan, the best chances are offered for lessening sickness in school children, because teacher, parents, family physician and inspectors cooperate.

SCHOOL FATIGUE AND BACKWARD CHILDREN.

In 1900, 16,000,000 children were enrolled in the common schools of our country. "These children are supposed to acquire knowledge and physical health." "The aim of an education must be the development of the fullest and soundest mental, moral and physical life of which the particular individual is capable," but a gain in knowledge at the expense of physical health is a loss. The opportunity for acquiring knowledge is the same for all school children, but not all children have the same capacity.

Many well founded complaints have been made of over-pressure in primary schools, and of undue pressure on backward scholars by keeping them in after school; by long home lessons, or by an injudicious use of emulation. The relative endurance of the sexes varies; girls do not gain much after puberty, as boys do, and about one girl in twenty is cbliged to give up her school life on account of ill-health.

The present-day native American girl of the middle class is the artificial product of advanced civilization. She is a bundle of nerves encased in a fragile frame. A prominent gynecologist has aptly said: "The American horse receives, on the average, better treatment than the young woman of America from the time of early girlhood until the age of development has passed. The stock breeder never forces the young animal during the period of development, realizing that it is the time when the greatest care should be taken; while American parents, especially of the middle

classes, with great ambition for their children, and the desire that they develop intellectually beyond their own standard, allow their heads to be crammed with knowledge so rapidly that the brain cannot assimilate it, and the result is that all strength of development is devoted to the brain, and physique finds expansion as best it can. New England furnishes the extreme type of the woman, supposedly more perfect than in any other section; intellectually above the average, but with a physique below par, with greatly reduced reproductive powers—all due to the forcing of study at the age of development."

Our school curriculum, as arranged by the average up-to-date pedagogue, is inclined to take cognizance of the socalled bright children endowed with a quick reaction time; but frequently the slow ones eventually show the greater mental strength and endurance and become the more intellectual and successful men and women. "The bright ones are like hothouse plants" and fall short of real maturity.

The days of brutally whipping children in school are gone; we are now refined and whip their brains to death. A correspondent of the *Lancet* wrote, in 1884: "If more evidence is necessary to prove the exhausting influence of school life, it would be obtained by a study of the group of head masters of our public schools. When they met at Wellington College in conference, I saw them photographed in group in front of the college, their faces

'Sicklied o'er with the pale cast of thought.'

Of some sixty masters I could pick out only three who looked ordinarily healthy." The same sad faces can be seen in the group pictures of the graduating classes of any public school in our cities.

In view of these facts the pediatrist may well call a halt and demand for our children less brain gymnastics and more fresh air and exercise. It is practicable to classify the children seen in a school by means of facts to be obtained from the teachers' reports and from medical inspection. About 20 per cent. of school children are found to be backward, solely as a result of some defect of the special senses; dullness due to eye defect or to defect in hearing. Another group is made up of neurotic children with low nutrition and mental dullness.

Backward children should have separate classrooms and special care, that they may develop to their maximum, and also that they may not be a hindrance and source of discouragement

to other and more precocious children; but the greatest obstacle to such a change would be the unwillingness of parents to admit that their children were backward or mentally deficient. Children with marked mental and physical abnormalities, developmental defects or stigmata should be segregated for special training.

The education of the feeble-minded lies essentially along the line of the development of the senses, which are not up to the normal standard. England, Germany and Belgium have each solved the difficulty in part by establishing special auxiliary classes, ungraded schools, where sub-normal children receive what is really private instruction based upon their individual needs. In our country about 6 per cent. are now cared for in special institutions, according to figures furnished by Dr. Louise F. Bryson. There are at present only sixteen states in the Union that provide custodial care for them. New York has two institutions; so has New Jersey. The other fourteen states that are up to date in this matter are California, Washington, Nebraska, Iowa, Kansas, Illinois, Minnesota, Ohio, Indiana, Kentucky, Maryland, Pennsylvania, Connecticut and Massachusetts. In Providence and Boston there are special schools for sub-normal children connected with the regular public school system. One of the duties of the physician, teacher and statesman is to find out the place of the feeble-minded. They have the same right to an education which will fit them for a sphere of usefulness that the blind have. The Widener Memorial Industrial Training School for Crippled Children is also a splendid example of a well directed humane charity. Although dull and deficient children are better detected by the teacher than by the physician, the classification of backward children, as to the intrinsic cause of the backwardness, and, eventually, the suggestion and application of the remedy for this backwardness, must be left to the physician.

BODILY TRAINING OF PUPILS AND SCHOOL BATHS.

The physician has a part in the encouragement of bodily training of pupils and of playground extension. "Inhibition, to sit still and hold muscular movements in check, is one of the first lessons of school life. Unnatural order should be dispensed with, and the children allowed all the physical activity compatible with the best results."

Playgrounds for Children.—Tsanoff, the secretary of the Culture Extension League, says: "The playgrounds stimulate and guide life in a way that nothing else can do. Their relation to

the development of character is very similar to that which the school bears toward the development of mind. In fact, the collective play of the children has a greater influence in forming habits of personal and social conduct of life than has school or even home instruction and advice. Many practical questions, for the solution of which we have been looking to the school, the home or the church, will be found belonging to the playground to solve. The time is coming when the playgrounds will be as seriously considered as the schools are to-day."

School Baths.—An important step was made by the Mayor of Göttingen, Germany, when he established shower or rain baths in connection with a large public school. A similar bath was established later in Weimar. Reports show that although very few children were at first disposed to take these warm shower baths, there were 200, or 75 per cent. of the whole number, visiting them daily after two months. The bathing is done during school hours, by a well arranged plan of relays, which does not interfere with instruction. "The provision for baths is of the greatest importance. It provides an opportunity for very poor children to enjoy the wholesome luxury of a bath, when otherwise they would not have one; besides, it is educational. The lesson of personal cleanliness is one of the fundamental principles in education."

The shower bath is the best and most sanitary for public bathing places, and probably of more importance to the community than public libraries.

An interesting and important question has been raised by the "Child-Labor Committee," which is best submitted by reproducing a letter recently received:

"My Dear Sir:

"The present child-labor law provides that before beginning work a child must not only be fourteen years of age, but must have acquired a certain minimum of education, which is about that of the normal twelve-year-old. This provision is being very carefully enforced in this city, and complaint has arisen against it in several quarters. It has been claimed that the law should provide rather that fourteen-year-olds who are deficient in schooling should be obliged to attend evening school.

"It is the belief of members of our committee that such a provision would be a very unfortunate substitute, and we should

very much value your opinion upon this point. Do you consider that fourteen-year-old children who are employed in factories nine hours a day can, without physical injury, attend school in the evening two hours longer? Is there any difference between boys and girls in this regard?

"Trusting that you may be willing to help our committee in preventing the amendment of the existing law, if the amendment indicated above seems to you unwise, I beg leave to remain,

"Yours very truly,

(Signed) "FRED S. HALL, *Secretary.*"

My reply was that, in my opinion, factory boys in good health might safely go to night school and thus escape to some extent the "loaferism" of the street. The factory girl should not as a rule be compelled to attend night school after nine hours' work.

INFANTS' AND CHILDREN'S HOSPITALS.

Medical opinion should discountenance the erection of large institutions in the city to be filled with chronic cases and minor ailments. City hospitals for children in particular are adapted only for acute cases of illness of a severe type. Country and sea shore sanitaria and floating hospitals are for the chronic cases. Hospitalism is a complex condition culminating in what is technically known as house infection. The latter is not confined to hospitals, but is a well-known phenomenon as observed in school dormitories, asylums, overcrowded and overheated private homes, lumber camps, soldiers' barracks, etc.

It has been observed by those in charge of children's hospitals everywhere that young babies, after convalescing from acute illness, will eventually, as a result of hospital life and surroundings, lose color, appetite and weight. We should therefore make it a rule to send children home as soon as their condition will permit, and particularly is this necessary when we suspect in a child a feeble resistance to infection by reason of prolonged malnutrition, in the socalled difficult feeding cases. Our regulations for fighting house infection in the Babies' Ward of the New York Post-Graduate Hospital are as follows:

(1) Strict cleanliness of premises and inmates. (2) Thorough ventilation. (3) Sufficient air space. (4) No overheating of wards. (5) Infants are not to be fed by the same hand that attends to the toilet. (6) Soiled linen is to be immediately

removed and disinfected. (7) Feeble children are toned up by a change of air (trip to Staten Island, omnibus ride through the Park and sojourn in the Sunbeam playroom and roof garden).

By adopting such precautions we believe that we have been very successful in preventing disease and in lowering our mortality.

EDUCATIONAL FEATURES IN PEDIATRICS.

Pediatrics is a department of general medicine, not a specialty; but special teaching in diseases peculiar to infancy and childhood is a necessity. Undergraduate teaching may be carried out by means of didactic lectures and hospital bedside instruction. Postgraduate teaching should be systematized on a strictly clinical basis; and physical diagnosis, laboratory work, dieto-therapy and hydrotherapy made a prominent feature in a course of pediatrics. All children's hospitals should be equipped as places for research and as teaching hospitals for undergraduates, postgraduates and nurses. Clinic rooms should be in direct connection with the out-patient department of hospitals, and matriculates should have the privilege of visiting dispensary patients in their homes.

In giving clinical instruction in pediatrics during the past fifteen years, I have adopted the following plan: The first forty minutes of the clinic hour are devoted to a more or less rapid demonstration of ten to fifteen sick children, who are placed on tables accessible to the matriculates, the balance of the hour being taken up by a discussion of the principles of treatment of important groups of diseases, such as pneumonia, eruptive fever, convulsions, difficult feeding cases, etc.; or a demonstration is given of stomach washing, bowel irrigation, spinal puncture, intubation, tracheotomy and other subjects. For facilitating direct bedside instruction I make use of a clinical instruction chart which was presented to the American Pediatric Society at a former meeting.

Conclusion.—I have dwelt but briefly upon the more important problems to be solved in matters of home, school and hospital hygiene. Other problems will come and go with time.

Pediatric societies should be the guardians of our children in all sanitary questions. They should be organized for the purpose of promoting the public understanding along these lines, and it is entirely within their province to offer suggestions to school and health boards on matters of sanitation.

In order to broaden the aim and scope of our Society work,

the programme of the annual meeting should be so altered and systematized as to suit or reflect the object of the Society.

Heretofore the clinical and laboratory experience of our members, with an occasional collective investigation, has been the order of business. It would seem feasible that our Annual Transactions might contain a running record of the world's progress in Pediatrics by presenting at each meeting a critical review of the world's progress during the year, on the following subjects:

Critical Review of the Year's Progress in (1) Home, School and Hospital Hygiene and Prophylaxis. (2) Diagnosis: Bacteriological, chemical and regional. (3) General Therapeutics, including nutrition, diet and hydrotherapy, photo-therapy, electro-therapy and mechano-therapy. (4) Surgical Therapeutics; Orthopedics.

Clinical contributions and original articles by the members could be grouped around these standards and would make up the balance of the programme. At each annual meeting, the council could designate the members who would be expected to furnish the critical review of progress for the next annual meeting, giving them a full year's time in which to collect material. In this manner our Transactions would contain a running record of the world's progress in the special department of medicine in which we labor.

I am also greatly in favor of prearranged discussions. We have often amused ourselves with uncertainties and called it discussion. In a scientific body, discussion should be the result of mature inquiry and express a clear statement. Writers of papers should be requested to furnish a brief synopsis of their communication to the members, who will then have time to formulate and arrange what they wish to say, and not weary the Society by digressive talk. We cannot afford to waste time, "for time is the stuff that life is made of."

But with all our shortcomings we have accomplished a great deal in the past fifteen years, and I am proud to be associated with men of national and international reputation. We have work behind us and more work before us. Let our philosophy be that of Shakespeare's Hamlet, who says:

"For some must watch,
While some must sleep;
So runs the world away."

CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS IN AN INFANT, WITH REPORT OF A CASE.

BY JOHN DORNING, M.D.,

New York.

On May 24, 1903, I saw, with Dr. Haring, of Tenafly, N. J., Baby H., *aet.* two months, whose history, obtained from the mother, is as follows:

The baby was born March 23, 1903, after a natural labor, of healthy parents, and is the fourth child, the remaining three being alive and healthy. Apparently healthy at birth and weighed eight and one-half pounds. Diet exclusively breast milk; but has, since birth, always vomited after nursing. The mother says the vomiting was excessive, very much more than with her other children. Sometimes she would nurse him three times in succession before she could lay him down without his vomiting the whole nursing. She never noticed mucus in the vomited material. The milk always returned fluid. The baby seemed to be able to retain water. The mother tried to nurse him every two hours but he would often sleep three to four hours between nursing. Frequently he would sleep from 6 P.M. to 6 A.M., and he was never awake more than once during the night. He seemed to be always sleepy.

He never cried, but would fret after vomiting until he nursed again. Bowels moved several times a day, always yellow in color and digested. On one or two occasions the stools were slightly green, but the yellow color returned after the administration of a teaspoonful of olive oil. The baby grew long, the face was plump and body not thin, but the legs and arms were always very emaciated. The abdomen was flat after long abstinence from food, but showed much distension after nursing.

On May 6th, when six weeks old, he vomited excessively, bringing up curds and mucus. The bowels became constipated and the urine scanty. After a few days he voided no urine at all for forty-eight hours, and the bowels did not move for several days. At this time a physician was called who prescribed salol for "infantile

grippe," also hot baths two or three times a day. Baby seemed to improve for a few days—kidneys and bowels both resuming their functions. On May 19th, had high fever; was some better next day, but continued to vomit and lose flesh; there was now mucus in the vomited matter.

I found a pale, extremely emaciated, very weak and listless infant. The abdomen was generally somewhat distended, but soft, tympanitic, and not tender on pressure. On palpation I felt a small mass in the epigastric region, about as large as a medium sized olive, just to the right of the median line. Examination of the chest was negative. Urine negative. Tongue was clean. I diagnosticated hypertrophic stenosis of the pylorus and gastric catarrh. Because of the low vitality of the baby operation was not entertained. I advised gastric lavage and, temporarily, nutrient enemata. I did not see the baby again until June 8th, when I performed an autopsy.

The nurse's and mother's record for the intervening period, states that until June 7th, when the child died, "the stomach had been washed out once or twice a day, bringing up milk and mucus each time; that he was nursed from four to eight times a day, not more than ten minutes at a time, and vomited from two to eight times a day. The vomited matter would usually consist of fluid milk, but sometimes it was curdled and mixed with mucus. For two days it was sour, at which time barley gruel and cream had been substituted for the breast milk. Nutrient enemata were occasionally given. The bowels were variable but there was no diarrhea. Medication consisted of the occasional administration of small doses of calomel, bismuth now and then and small quantities of brandy at varying intervals. On the night of June 5th, the abdomen became much swollen and the baby very fretful. Irrigation of the bowels relieved the distension and afforded great relief. At 2 P.M., June 7th, the baby became unconscious while nursing and died two hours later," at the age of two and a half months.

AUTOPSY June 8th, 4 P.M. Body extremely emaciated. Rigor mortis not marked. Liver, spleen and kidneys showed nothing abnormal. Stomach distended. Pylorus much thickened and hard. Duodenum, one inch beyond pylorus, deeply congested over an area of an inch-and-a-half, resembling a gut that had recently been released from strangulation. The stomach contained about two ounces of milk; a large amount of thick and very tenacious

mucus adhered to the mucous membrane and extended into the pylorus, blocking up its lumen. The mucous membrane of the stomach was pale and showed no ulceration. The wall of the stomach presented a decided thickening toward the pyloric extremity. The pylorus itself showed a pronounced thickening of its walls and a narrowing of its lumen, permitting the passage of a probe 2 mm. in diameter. Its lining membrane was thrown into longitudinal folds. The mucous membrane at the seat of the apparent strangulation was very deeply congested.

Report of examination, made by Dr. John H. Larkin, of the Pathological Laboratory of Columbia University: "The specimen for examination consists of stomach of infant with intestines attached. It is moderately enlarged; consistence is very firm; and examination of its mucous membrane shows partial loss of normal corrugations, the mucous membrane being smooth and without evident signs of ulceration. The fundus and pyloric portion show marked thickening. This portion is funnel shaped, the

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See Frontispiece for Illustration

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greater curvature, at its greater diameter, 5 cm. On longitudinal section of the pylorus along the greater curvature, the pylorus measures from its tip in the duodenum to that part where the pylorus suddenly ends and joins the general stomach wall, 3 cm. The thickness of the pylorus behind the gastroduodenal valve is 5 mm. Passing backward at a point 5 cm. from the duodenal tip the stomach wall measures 3 mm. From this point the stomach wall gradually thins out till we come to a point at the greatest diameter of the stomach along the larger curvature where the stomach wall measures 2 mm.

"The mucous membrane at pylorus is markedly thickened and folded longitudinally. There are no evidences of ulceration of the mucosa.

"*Microscopical Examination.*—Portions were taken as follows: (1) A longitudinal section of thickened pylorus including part of the duodenum, 2 cm. (2) A cross section of pylorus. (3) A cross section at fundus. (4) A section at greater curvature near the anterior surface.

"The microscopical picture is one of a diffuse fibrous thickening of the submucosa and an extensive hyperplasia of the circular muscular fibers at this point. The longitudinal muscular fibers also show moderate thickening. The submucous tissue forms a very dense fibrous tissue which is devoid of cells and shows but few blood vessels. The circular muscular fibers are arranged in bundles and separated by dense fibrous tissue strands which are continuous with those of the submucosa.

"The sections at the fundus show essentially the same picture of thickened submucosa but in less degree the thickening of the circular fibers.

"Examination of the mucous membrane at pylorus shows the glandular elements intact and normal. The mucous membrane of the duodenum was bile stained and its glands were necrotic, otherwise there is no pathological change in this part of the specimen. The mucous membrane at fundus and at greater curvature shows loss of its glandular arrangement. The cells have desquamated so that the picture is one of a great many larger and smaller cells aggregated together, in place of well defined glands.

"The anatomical diagnosis is, therefore, Stenosis of pylorus from fibrous thickening of the submucosa and hyperplasia of the circular muscular coat."

In recording this case of congenital hypertrophic stenosis of the pylorus, there is added to the rapidly increasing list another unmistakable instance of this affection, the diagnosis of which was verified by autopsy.

The first recorded case, that we know of, was by Williamson,[1] in 1841. In 1842 Davorski[2] reported a second case. There is then no further mention of this malady until 1888, when Hirschsprung reports 2 cases. There are now on record about 60 cases in all. The affection, therefore, while not common, cannot be said to be rare. It is a remarkable fact that most observers after having seen a single case and thus had their attention directed to the affection have subsequently come across one or more additional ones.

A study of the literature of this affection would lead one to infer that there are either two distinct pathological conditions with similar clinical manifestations, one of which is amenable to dietetic management and the other, unless relieved by surgical operation, progresses to a fatal termination; or, that there are simply varying grades, or perhaps different phases of one and the same pyloric lesion. The published pathological findings could, thus

far, be interpreted to favor either view. In nearly every reported case, in which an autopsy was obtained, hypertrophy of the circular muscular coat is noted, while in most of them there is also hyperplasia of the submucosa and in a few some thickening of the mucosa. The pathological condition in the reported cases of recovery can, of course, only be conjectured.

The pathogenesis of this affection is still a matter of speculation. It is generally conceded that it originates during intrauterine life. Thomson's[17] theory that it is "a functional disorder of the nerves of the stomach and pylorus," causing spasm and secondary hypertrophy of the pyloric structures is not altogether satisfactory, in that while it may, in a measure, account for the muscular hypertrophy, it does not explain the increase in the submucous connective tissue, present in so many of these cases.

The condition is also thought to be simply a prenatal overdevelopment of the pyloric structures, or, in other words, an embryological malformation with probably secondary spasm of the muscular coat. It must be confessed that, at present, we know very little about its pathogenesis or etiology. The possible existence of a syphilitic element in this condition should not be forgotten, even though the immediate history be negative.

The diagnostic symptoms of hypertrophic stenosis of the pylorus are persistent, and apparently causeless, vomiting, progressive wasting and a palpable pylorus. The vomiting is usually unaccompanied by nausea; it may, or may not, be forcible in character and may follow each feeding or may occur after every third, fourth or fifth feeding, in which case, the quantity is generally large. All the food ingested is not necessarily regurgitated, for some of it undoubtedly passes through the pylorus even though its lumen be exceedingly small. The vomited material may be in a fluid state or curdled; it is sometimes mixed with mucus and may show a normal, diminished or absent acidity according to the absence or presence of a complicating involvement of the gastric mucous membrane and dilatation.

The absence of bile in the vomitus would ordinarily indicate the obstruction to be above the entrance of the common bile duct into the intestine. Its presence, however, would not exclude pyloric stenosis, for Schwyzer[16] and Saunders[40] each report a case in which bile was mixed with the vomited matter and caused some uncertainty in diagnosis. In cases of moderate stenosis in which, perhaps, muscular spasm is a prominent factor, the gastric

symptoms, though sometimes rather pronounced, are generally not very urgent in character, and improve and even seem to entirely subside under dietetic treatment.

It is unnecessary to remark that the wasting is due to insufficient nutrition. It would not be unreasonable to believe that many cases of socalled marasmus or infantile vomiting are really instances of pyloric stenosis.

The presence of a palpable tumor in the region of the pylorus, in conjunction with the previously mentioned symptoms, would establish the diagnosis beyond a reasonable doubt. As it is not always possible to detect, with the palpating fingers, small masses in the upper abdominal zone, its absence would not altogether negative the diagnosis. Where the patient's condition would warrant it, an examination under anesthesia might disclose the presence of a thickened pylorus where it could not otherwise be detected.

The vomiting of abnormally large quantities of food, and a fulness in the epigastric and upper umbilical regions, with perhaps the demonstration of a peristaltic wave, would indicate a complicating dilatation of the stomach.

The tongue is frequently clean and the breath free from odor, unless fermentation be present. There is constipation in many cases. The symptoms, then, of congenital pyloric stenosis are practically those of pyloric obstruction, and other lesions, such as atresia, polypi and adjacent neoplasms or cicatricial tissue occluding the duodenal extremity of the stomach are so rare in infancy that when symptoms of persistent pyloric obstruction are in evidence this condition should at least be strongly suspected if not, with good reason, be considered to actually exist.

Every case is not necessarily immediately fatal. The blocking up of the lumen of the pylorus by thick tough mucus, as was observed in the subject of this report, may precipitate a fatal termination in a case that, otherwise, might continue on indefinitely. A small number of cases, in some of which the diagnosis may, perhaps, be questioned, with fairly well marked symptoms, are reported to have yielded to dietetic treatment and a few have apparently recovered after operation. Some of these patients undoubtedly live to an advanced age, all the while suffering more or less with dyspeptic symptoms. There are thirty-one autopsies published by Maier in which stenosis of the pylorus was found. The two youngest of these subjects were twelve and sixteen years

respectively, the oldest was eighty, and the ages of the others ranged between twenty and forty years. In some of the cases the clinical history dated back to childhood. There being no evidence of any pathological process likely to cause the lesion the cases were considered congenital. Landerer reports a case of marasmus in a man forty-five years of age, who had from infancy suffered from dyspeptic symptoms. The autopsy revealed a greatly dilated stomach with a pylorus having a lumen of only 2 mm. in diameter, but no hypertrophy of its walls. For about fourteen years I have had under observation a patient, now in his seventeenth year, who has been vomiting since he was three months old. At such times as his diet can be restricted to fluid nutriment, not including milk, there is an improvement in his general condition. At present he weighs but fifty-seven pounds and measures fifty-one and a half inches in height. He is thin and under-developed.

It is impossible with our present knowledge to predict with any degree of certainty what the future of these cases will be, whether operated on, or not. We know very little of the after history of gastro-intestinal operations in early life. Nicoll[39] mentions a case in which pyloroplasty was performed and the child eight months later was alive and thriving. Instances are published of recontraction of the pylorus after the operation of dilatation, and of occlusion of the new opening made by gastroenterostomy. I have in mind an unpublished autopsy in a very young infant performed several weeks after a gastroenterostomy in which the gastric mucosa, but not that of the intestine, was found to be so contracted as to permit the passage of only a very fine probe.

Nevertheless, in cases that are intractable to dietetic treatment operation alone offers any promise of relief and it should not be too long delayed.

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DISCUSSION.

DR. NORTHRUP.—I should like in general to encourage a healthy scepticism in the Society regarding such reports. Of course this case has been carefully studied. In the next few years there will be an epidemic of such cases as a result of a few cases reported. Many of them will be mistaken observations.

DR. WEST.—In looking up the subject before reporting my case to the Society last year I found only 4 recorded cases of true hypertrophic stenosis of the pylorus. The distinction is made by but few writers between cases of congenital gastric spasm, with hypertrophy of the circular muscular layer, and cases of true stenosis in which there is hyperplasia of the epithelium, increase of connective tissue in the submucosa and of fibrous tissue in the muscular coat, with hypertrophy of both muscular layers. An illustration of this is found in an article published in the ARCHIVES OF PEDIATRICS some time since in which a case of each kind is reported.

That the spasm of the sphincter has considerable to do with the termination of the trouble, even in the latter cases, is shown by the fact that, postmortem, the pylorus permits the passage of a good-sized probe.

With regard to Dr. Dorning's statement that the first case of this kind was reported in 1841, attention ought to be called to the case published by Dr. Hezekiah Beardsley, in 1788, and republished by Dr. Osler in the ARCHIVES OF PEDIATRICS for May, 1903. While no microscopical examination was made of the case it appears to be a true hypertrophic stenosis of the pylorus in a boy six years old, who had been sick since birth.

DR. MORSE.—A year ago I occupied somewhat the same position as Dr. Northrup in his scepticism when such cases were reported. During the past year, however, I have seen 2 cases in which the symptoms corresponded accurately to the descriptions given of those of hypertrophy and stenosis of the pylorus. They were fed on all sorts of food, including breast milk. There was

obstinate constipation and progressive loss of weight. In 1 case there was a palpable tumor and visible peristalsis. Both infants were operated on. In both cases an enlarged mass was found at the pylorus. Gastroenterostomy was performed in each case, and death resulted in both cases, in one after thirty-six hours, in the other after a number of days. No autopsy or microscopical examination was made in either case.

DR. HOLT.—It would be a pity to allow the impression to prevail from this discussion that any doubt existed regarding the occurrence of this condition. I have recently seen 2 cases which presented many of the features of the case reported by Dr. Dorning. One child was six weeks old and began to vomit at the age of two weeks. It would take food in the morning and retain it, and again at eleven, at two and at five o'clock food was retained, until about six o'clock each afternoon when almost the entire food taken for the day would be vomited. In this infant the stomach was dilated to the size of 8 ounces. The case was a perfectly typical one. The child was operated upon, but unsuccessfully, and the microscopical examination confirmed the diagnosis. Another case seen in hospital practice this year gave a similar clinical picture and the same results from a microscopical examination. I do not believe cases of hypertrophic stenosis of the pylorus are common, but they certainly are not so very rare.

DR. ROTCH.—I have seen a case which presented the symptoms described of pyloric stenosis, but the autopsy showed nothing of the kind; instead, there was a dilatation of the cardiac end of the esophagus. The symptoms presented were very similar to those of stenosis of the pylorus.

DR. CAILLÉ.—I think that the greatest interest in these cases of stenosis of the pylorus is centered in the diagnosis. The clinical picture of obstruction at the pylorus is occasionally seen. In the absence of palpable tumor this condition is one in which it is most difficult to make a correct diagnosis. The diagnostic features are still to be studied.

DR. DORNING.—I was somewhat sceptical regarding some of the reported cases of congenital hypertrophic pyloric stenosis. I think the specimen presented will speak for itself, as it shows a great disproportion between the muscular and mucous coats, and the development of fibrous tissue in the submucosa. I agree with Dr. Caillé that it is almost impossible to make an exact diagnosis in the absence of a palpable tumor; if a tumor cannot be made out the diagnosis must still remain in doubt.

PYLORIC STENOSIS IN INFANCY. REPORT OF A CASE; GASTROENTEROSTOMY; RECOVERY.

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AND

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Since the attention of the medical profession was directed to the clinical features resulting from a stenosis of the pylorus, a large number of cases have been recorded, and the condition is no longer looked upon as an anatomical curiosity.

The following case is of more than usual interest on account of its successful termination after surgical interference:

Esther S., born January 31, 1902, and admitted to St. Margaret's House, January 3, 1903, aged eleven months. She is the second child of Russian Jew parents, whose limited knowledge of English makes a complete history difficult. Both parents are strong and healthy, as is the other child, who is thirteen months older. The pregnancy and labor were uneventful, and the child weighed ten pounds and a half at birth. The mother nursed the baby at the breast exclusively, until the admission to the hospital, although she confessed to having given her a few things to eat from the table after the eighth month. The child gained rapidly in weight during the first three or four months. The exact date when the vomiting commenced is not known. It is supposed to have begun about the fifth or sixth month, but was not regarded seriously, as the mother had been told it was a healthy sign to have the baby spit up after feeding. After this time a little food came up after each nursing.

About the end of November she had an attack of bronchopneumonia, which was followed by empyema. This was aspirated three weeks before admission, and on account of the vomiting and poor physical condition, she was brought to St. Margaret's House.

On admission, the child weighed 15 pounds, 8 ounces. The body was emaciated, and the skin and mucous membranes pale. The nostrils dilated with each respiration, but there was no cyanosis. The breathing was labored, and the respirations varied from 34 to 54 a minute. The temperature ranged from 99 to

101.5, and the pulse was strong and regular, numbering from 100 to 140 beats a minute. The lower border of the liver and the spleen were palpable. The left lung was slightly bulging and flat on percussion. The apex beat was one inch to the right of the nipple line in the fifth interspace. The breath sounds could be plainly heard over the area of dullness. The examination of the blood showed 35 per cent. hemoglobin, 21,700 leukocytes, 5,144,000 red corpuscles. Smears from the pus showed streptococci. A rubber drainage tube was inserted on January 23 and removed February 10, when the lung symptoms cleared up.

The most unpleasant feature of the case was the persistent vomiting. A portion of each feeding would be rejected from ten to thirty minutes after each bottle. Very often she would hold the regurgitated food in her mouth for several minutes, and again attempt to swallow it. Lavage was faithfully kept up twice a day for three weeks, without any influence on the vomiting, and for two weeks all the food was administered through a stomach tube without success. The food itself was changed in composition and in the amount and frequency of feeding with no effect on the vomiting. Fluids were withheld and only semi-solid nourishment given, and the food was predigested without helping the condition. Many vaunted medicinal remedies were administered without benefit. The child seemed ravenously hungry, and always took the food with great eagerness.

In five months she lost 3 pounds and 6 ounces, and was greatly emaciated. The stomach contents were examined a number of times, but no free hydrochloric acid or lactic acid was detected. The vomiting material was always curdled and sour, but it was never tinged with bile. The bowels were always very constipated, and high enemas were given daily. The movements themselves were hard and scybalous. The abdomen was sunken and the peristaltic movements plainly seen. The stomach was dilated and extended down to about the level of the umbilicus. Repeated examinations failed to detect a tumor of the pylorus.

The diagnosis of pyloric stenosis was based on the incessant vomiting, dilated stomach, constipation, absence of bile in the vomited matter, and steady loss in weight. In view of the futile attempts to alleviate the condition by medical treatment and the apparently hopeless state of the child, permission was granted for an operation. The operation was performed on June 8, 1903, under ether narcosis, by Dr. Elting.

A median incision 8 cm. long was made from the ensiform

process downward. The abdominal wall was extremely thin, the abdominal muscles atrophic. Immediately on incising the peritoneum, the greatly dilated stomach presented in the wound. Examination of the pylorus showed it to be very firm and thickened for a distance of 2½ cm. The transverse diameter of the pylorus was about 2 cm. The lumen appeared to be greatly reduced and the stenosis pronounced. There were no adhesions about the pylorus, and no evidence of an inflammatory process, the peritoneum being smooth and glistening. The stomach was at least four times its normal size. The walls were extremely thin. A loop of the jejunum was drawn up, and at a point about 40 cm. from the duodenum an anterior gastroenterostomy was performed after the method of Kocher, according to which a spur is made in the jejunum and the intestine is attached transversely to the lowest part of the anterior wall of the stomach. Catgut was used to unite the mucous surfaces, and over this two layers of continuous silk sutures.

The introduction of the sutures into the stomach and bowel was rather difficult, because of the paper-like thinness of the walls of these viscera. The operation was completed with a minimum of handling of the stomach and intestine in order to avoid shock. The peritoneum was closed with continuous catgut. The fascia and muscle were closed with interrupted catgut, and the skin with subcutaneous catgut. The time of the operation was forty-five minutes. The patient appeared to be in as good condition after the operation as before, which, however, was decidedly bad. During the night previous to the operation, the nurse in attendance on two or three occasions thought the child dying, but it was revived by means of injections of brandy and strychnin. The wound healed per primum, and has remained firm and strong ever since.

Subsequent History.—Shortly after the operation she vomited blood. On June 9 was given a bottle of peptonized milk and barley, which was retained. She vomited some bile during the day. The food was gradually increased, and a number of bottles were retained, although she would vomit several times a day. A soft yellow stool was returned after an enema on June 14. The first soft spontaneous movement was on June 18. The child improved steadily, and after four weeks the vomiting occurred only occasionally. The food was increased gradually, and the child gained steadily in weight, and left St. Margaret's on December 17, weighing 22½ pounds. Nine months later she weighed 30 pounds, and seemed perfectly well. The weight chart shows the rapid and steady gain after the operation.

Stenosis of the pylorus in infants is not a very rare or unusual condition. Cautley and Dent, in an essay read before the Medico Chirurgical Society, of London, in 1902, appended a bibliography of 109 references.

They collected 69 cases, and in 19 of these an operation had been performed. We have searched the literature up to August, 1904, and have found 66 cases in infants under twelve months in which the diagnosis was confirmed by autopsy; 12 cases in

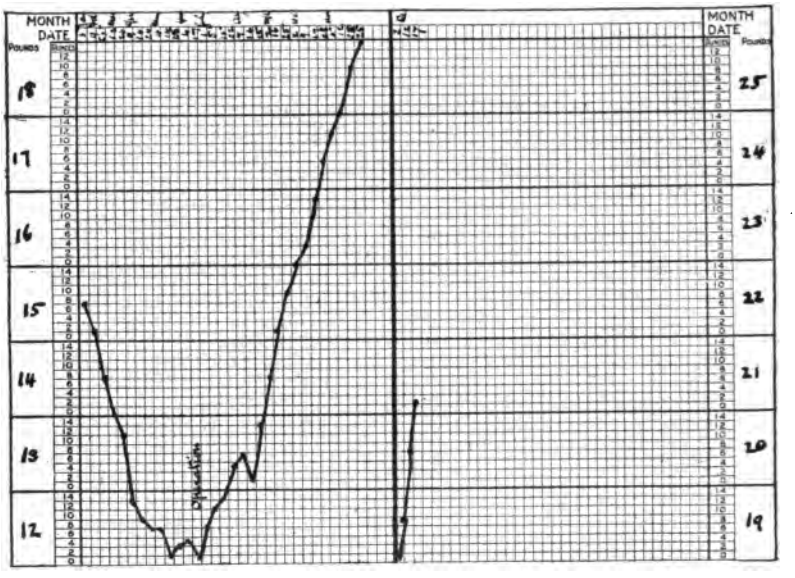

CHART SHOWING DECREASE IN WEIGHT BEFORE, AND INCREASE IN WEIGHT AFTER, THE OPERATION.

which no autopsy was reported; 27 cases diagnosed as pyloric stenosis, but ending in recovery, and 40 cases in which a surgical operation had been performed, making 145 cases in all. These cases are nearly all reported by specialists, so that it is fair to presume that the oft-misused and misunderstood diagnosis of marasmus has been given to describe the condition without any recognition of the underlying cause.

We propose to discuss only briefly the pathogenesis of this disease. In order to better appreciate this condition, we will review a few of the theories that have been advanced, and consider certain points in the anatomy of the parts involved.

The stomach has only slight powers of absorption or assimilation, and is more a motor than a digestive organ. The muscular

tissue is made up of three sets of fibres, all of which extend to the pylorus. The stomach proper has also a secretory function, but the work of the pylorus is chiefly motor.

The circular set of muscular fibres are greatly increased at the pyloric orifice, forming what is called the sphincter pylori. The cardiac end has no special sphincter, and gas and liquids can readily pass through it during digestion. The mucous membrane at the pylorus is thicker and forms a fold known as the pyloric valve. There are traces at the pars plyori of a second gastric chamber in the bulging called the antrum pylori. The gastric cells and tubules in the neighborhood of the pylorus differ in construction and function from those located elsewhere in the stomach.

The pyloric region is essentially one of considerable structural variability. The pneumogastric nerves supply both motor and sensory filaments. Bechterew has shown that by irritating one branch of the pneumogastric, without interfering with the others, the cardiac end of the stomach will dilate, while the stomach and the pylorus remain contracted. This proves that each portion of the stomach has its special nerve filament. The splanchnic nerves connect the stomach with the sympathetic nervous system. The pylorus has the power within itself of contraction or dilation, and the contraction is always increased by the ingestion of food.

A stenosis of the pylorus may occur, according to some authors, as a congenital malformation. The circular muscular fibres are excessively developed, producing a violent contraction of the sphincter with the resulting retention and subsequent vomiting. In other words, the claim is made that this is a primary condition where "Nature, in her extreme anxiety to provide an efficient pyloric sphincter, has over-exerted herself, and has produced too great a quantity of muscular tissue." Flynn points out the similarity of the pylorus with the gastric mill in insects and crustacea and with the gizzard in birds. He thinks that hypertrophic stenosis is a reversion to the edentate pylorus as seen in the armadillo, or the great ant eater, and quotes Darwin as saying that "certain structures regularly occurring in the lower members of man's own class occasionally make their appearance in him, though not found in the human embryo; or, if normally present in the human embryo, they become abnormally developed, although in a manner which is normal in the lower members of the group."

If the hypertrophy were congenital and primary the vomiting ought to commence soon after birth. As a matter of fact, this

occurs only rarely, and in the large majority of cases the first vomiting is not noticed until the child is several weeks old. In our case the vomiting did not begin until the child was five or six months old. This explanation cannot account for all the cases, as Ballantyne, whose knowledge of fetal pathology is very extensive, has never seen an hypertrophied pylorus in the fetus.

Pfaundler advanced a theory about six years ago in which he denies the existence of any hypertrophy, and claims the entire symptom complex is produced by a spasm of the pylorus without any hypertrophy or stenosis. He based his conclusions on the result of postmortem studies on the stomach of infants. He frequently found that the pylorus was firmly contracted and presented all the appearances of pyloric stenosis. This condition he termed a functional *systolic* stomach in contradistinction to the relaxed or *diastolic* stomach. Undoubtedly, many cases of pylorospasm or functional spastic stenosis without hypertrophy exist and include many of those reported as pyloric stenosis which make a good recovery. That this does not explain the severe and fatal cases is shown in the reports of the 86 cases in which an autopsy was performed, where the pylorus was invariably found greatly thickened. Accurate measurements and histological sections were made in many of these cases. A functional spasm of the pylorus would relax under anesthesia, but this was not observed in any of the operated cases.

The most plausible view, and one held by many authors, is that the hypertrophy is secondary and a result of overaction on the part of the pylorus. Such a condition is sometimes found in adults as a result of a reflex spasm occurring in ulcus ventriculi or hyperacidity. In infancy the condition is more likely due to increased irritability of the nervous mechanism or hyperacidity,* and from the overaction of the pylorus the muscular fibres are increased and hypertrophied and the lumen diminished. Larkin showed in the microscopical examination of the pylorus in his case that there was not only a marked hypertrophy of the muscular fibres, but also an increase in the connective tissue between the muscular fibres. John Hunter pointed out many years ago that hypertrophy from repeated forcible contractions is common to all muscles, but is greater in involuntary than in voluntary muscles. This explanation coincides with the clinical history of

* Examinations of one hundred and nineteen stomach contents made in the summer of 1903, showed a total acidity ranging from two to thirty-six and free hydrochloric acid in thirty-five cases. (Shaw.)

most of all the cases where the vomiting begins several weeks after birth and grows progressively worse.

The chief symptom of this condition is the vomiting. It occurs from ten to fifteen minutes after taking the food. Diminishing the quantity of the food or changing its strength or character has no effect. Most of the reported cases have been in breast-fed babies. It resists treatment by lavage and medicinal remedies. A characteristic feature is that the vomited material is never tinged with bile. In the majority of cases no free hydrochloric acid was found, but Freund found it present in 3 of his cases. One of these contained free hydrochloric acid before the operation and none was detected after the operation. The abdomen in these cases is never distended as is seen so frequently in chronic indigestion, but is sunken and relaxed. This permits the peristaltic movements of the stomach to become visible. The stomach becomes greatly dilated, and its outlines can be clearly seen through the thin abdominal walls. In many cases a distinct enlargement of the pylorus can be detected on palpation.

Constipation invariably results from curtailment of the food and fluid supply to the intestines. The movements are small in amount and are hard and scybalous. The urine is also diminished in quantity. The uncontrollable, long-continued vomiting, without any symptoms of dyspepsia or indigestion, dilated stomach, emaciation, lax and sunken abdominal walls, obstinate constipation, and, perhaps, an enlarged pylorus, produce a symptom complex not difficult to recognize.

The prognosis is grave. Only 27 out of 104 cases treated medically recovered, which gives a mortality of 72 per cent. Seventeen out of the 39 cases treated surgically recovered, giving a mortality of 56 per cent. It would seem from the statistics that the surgical treatment gives more chance of recovery than purely medical treatment.

The medical treatment should be commenced as early as possible in order to control the spasm before the hypertrophy results. The food should be given at frequent intervals and the stomach washed out once or twice a day. Changes can be made in the composition of the food, but experiments along this line are not always beneficial. A breast-fed infant should not be entirely deprived of its natural nourishment, but a "two-milk" diet can be instituted with a few bottles of artificial food. As some cases of pylorospasm are due to hyperacidity, it might be well to follow the advice of Knöpfelmacher and administer small amounts of

undiluted cow's milk. To this could be added citrate of soda as recommended by Poynton. Small doses of cocain before each bottle may give temporary help, but both the medicinal and the dietetic treatment will often prove unsatisfactory. High rectal irrigations of normal salt solution should be given daily in order to supply fluid to the tissues and to remove the hard constipated feces. Surgical treatment should be instituted when the diagnosis has been verified, so that the child will not be too debilitated and emaciated to withstand the shock.

That medical treatment of cases of pyloric stenosis in children has been unsatisfactory is shown by the relatively large number of autopsies upon cases of this description reported in the literature. Most pediatrists are agreed that in the majority of the cases of pyloric stenosis in the infant surgical treatment is indicated. The first operation for the relief of this condition was performed by Stern, June 3, 1897. In this case a gastroenterostomy was done and the patient died. The first successful operation for this condition, also a gastroenterostomy, was performed by Löbker, July 25, 1898. We have collected from the literature reports of forty operations upon infants with pyloric stenosis. Of these cases one was simply an exploratory incision, at which no attempt was made to relieve the condition, and the patient died. Another was a pylorectomy, and this patient also died. Of the remaining 38 cases 19 were gastroenterostomies, of which 7 recovered and 12 died, a mortality of 63 per cent.; 8 were pyloroplasties, of which 5 recovered and 3 died, a mortality of 38 per cent.; 11 were divulsions of the pylorus, the socalled "Loreta operation," of which 5 recovered and 6 died, a mortality of 55 per cent.

From the above, it appears that four different operations have been proposed and practised—pylorectomy, pyloroplasty, divulsion of the pylorus and gastroenterostomy. Pylorectomy can, without question, be excluded from the list of possible and practicable operations in such a condition; first, because there is no indication for the excision of the pylorus, and secondly, because in such weakened children an operation of this magnitude is almost certain to prove fatal. Of the three remaining methods of operation, the two which appear most rational and feasible are those of pyloroplasty and gastroenterostomy. Divulsion of the pylorus or, as it is more commonly known, "Loreta's operation," may offer temporary relief, but there is always a strong possibility of a recurrence of the stenosis and the danger attending this operation is quite as great as that of either pyloroplasty or gastroenter-

ostomy. "Loreta's operation" upon the adult has not been of great value, and is today done only infrequently, and the same arguments advanced against its practicability in the adult would, we think, hold equally well in the infant.

Pyloroplasty, theoretically and practically, appears to have much in its favor, especially from the relative ease of its performance in many cases. There are, however, certain objections which can be raised against it, especially the possibility of the development of adhesions about the pylorus which might subsequently seriously interfere with its function. Furthermore, we believe that the technical difficulties as well as the dangers of this operation are quite as great as are those of gastroenterostomy. In our judgment there is more to be said in favor of gastroenterostomy than of any other operation yet proposed for the cure of this condition. In the first place, it relieves the pylorus of the irritation of the food and gastric juices, which appear in some instances to be an important factor in the production of the stenosis. This irritation removed, it is fair to assume that the lumen of the pylorus would become increased and more or less of a normal function allowed. We would emphasize especially the necessity of avoiding handling the stomach or intestines more than is absolutely necessary, because this undoubtedly contributes largely to the shock of the operation. To this end it would seem more advisable to employ a method of anterior gastroenterostomy, which can be easily and quickly performed with a very minimum of handling of the abdominal viscera, and practically as quickly as pyloroplasty. It is also advisable to administer nourishment by the mouth very soon after the operation, for these patients are always greatly reduced and in immediate need of nourishment. Experiment and practice have shown that this can be safely done without danger to the integrity of the anastomosis and with certain benefit to the patient.

In conclusion, we believe that the following generalizations can be made:

(1) That pyloric stenosis in the infant is of much more frequent occurrence than is commonly supposed.

(2) That medical treatment of these cases has proven uncertain and unsatisfactory.

(3). That surgical interference should be practised before the strength and vitality of the patients have become seriously reduced.

(4) That either pyloroplasty or anterior gastroenterostomy are the operations of choice.

LIST OF REPORTED CASES OF PYLORIC STENOSIS IN INFANTS.

MEDICAL CASES.

CASES IN WHICH AN AUTOPSY WAS MADE WHICH CONFIRMED THE DIAGNOSIS.

Author.	*No. of Cases.*	*Date.*	*References.*
Beardsley	1	1788	ARCHIVES OF PEDIATRICS, 1903.
Williamson	1	1841	London and Edinburgh Monthly Journal of Medicine.
Davoski	1	1842	Casper's Wochenschrift f. d. ges. Heilkunde.
Peden	1	1889	Transactions Glasgow Pathological and Clinical Association, 1892.
Hirschsprung	2	1888	Jahrbuch für Kinderkrankheiten, 1888 and 1901.
Henschel	3	1891	Archiv. für Kinderkeilkunde.
Pitt	1	1892	Transactions Pathological Society of London.
Tilger	1	1893	Virchow's Archives
Gran	4	1896	Jahrbuch für Kinderkrankheiten.
Finkelstein	4	1896 1897	Jahrbuch für Kinderkrankheiten. Berliner klinische Wochenschrift.
Schwyzer	2	1896–7	New Yorker med. Monatschrift.
Ashby	2	1897	ARCHIVES OF PEDIATRICS.
De Brun Kop	1	1897	British Medical Journal.
Fenwick	2	1897	Disorders of Digestion, p. 315.
Stern	1	1890	Deutsche med. Wochenschrift.
Rolleston	1	1898	British Medical Journal.
Batten	1	1899	London Lancet.
Still	3	1899	Transactions Pathological Society of London.
Pritchard	1	1900	ARCHIVES OF PEDIATRICS.
Fisher	1	1900	British Medical Journal.
Larkin	1	1901	Proceedings of the New York Pathological Society.
Schultze	1	1901	*Ibid.*
Thomson	8	1902	London Lancet.
Saunders	1	1902	ARCHIVES OF PEDIATRICS.
Nordgren	2	1902	Eira. ref. Monatschrift für Kinderheilkunde.
Riviere	1	1902	London Lancet.
Selter	2	1902	Monatschrift für Kinderheilkunde.
Freund	3	1903	Mitt. aus den Grenz. der Med. u. Chir.
West	1	1903	ARCHIVES OF PEDIATRICS.
Shaw	1	1903	Brooklyn Medical Journal.
Cautley	6	1904	British Journal for Children's Diseases.
Bernhein	1	1904	Korresp für Schweiz. Aerzte.
Schotten	1	1904	Sammlung Klinische Vorträge, Leipzig.
Cleveland	1	1904	British Medical Journal.
Fisher, Neild	1	1904	Bristol Medico-Chirurgical Journal.
Dorning	1	1904	ARCHIVES OF PEDIATRICS.
	66		

FATAL CASES IN WHICH NO AUTOPSY WAS REPORTED.

Henschel	2	1888	Jahrbuch für Kinderkrankheiten.
Finkelstein	3	1896	Jahrbuch für Kinderkrankheiten.
Stern	2	1898	Deutsche med. Wochenschrift.
Thomson	3	1902	London Lancet.
Salmon	1	1903	Monatschrift für Kinderheilkunde.
Cariot	1	1903	Gaz. des Mal. Infants.
	12		

CASES DIAGNOSED AS PYLORIC STENOSIS, BUT ENDING IN RECOVERY.

Author.	*No. of Cases.*	*Date.*	*References.*
Finkelstein	3	1896	Jahrbuch für Kinderkrankheiten.
Senator	1	1897	Berliner klin. Wochenschrift.
Still	1	1895	Transactions Pathological Society of London.
Coates	1	1900	ARCHIVES OF PEDIATRICS.
Knöpfelmacher	1	1901	Wiener klin. Wochenschrift.
Saunders	4	1902	ARCHIVES OF PEDIATRICS.
Burghard	2	1902	London Lancet.
Gardner	1	1903	London Lancet.
Koplik	3	1903	Diseases of Infancy and Childhood.
Schotten	3	1904	Sammlung Klinische Vorträge, Leipzig.
Variot	2	1903	Gaz. des Hôp.
Ibrahim	4	1903	Jahrbuch für Kinderheilkunde.
Conitzer	1	1904	Münchner med. Wochenschrift.
Stamm	2	1904	Münchner med. Wochenschrift.
	27		

SURGICAL CASES.

GASTROENTEROSTOMY.

Physician.	Surgeon.	Age.	Died	Time After Operation.	Recovered.	References.
Stern		5 weeks	1	24 hours		Deutscher med. Wochenschrift, 1897.
Adler	Meyer		1			Medical Record, 1898.
Meltzer	"		1			Medical Record, 1898.
Abel	Kehr	8 "			1	Münchner med. Wochenschrift, 1899.
Thomson	Stiles	5 "	1	24 "		British Medical Journal, 1902, p. 1,874.
Monnier	Fritzsche				1	Dissert. Zurich, 1900.
	Löbker		1			Vers. deutsch. Gesell. f. Chir., 1900.
	"				1	Vers. deutsch. Gesell. f. Chir., 1900.
	K hr				1	Vers. deutsch. Gesell. f. Chir., 1900.
	Trautenroth	5 "			1	Mitt. aus den Grenz. der Med. u. Chir., 1902
Mackay	Mackay	4 months	1	36 "		Intercolonial Medical Journal, 1903.
Ibrahim	Jordon		1			Münchner med. Wochenschrift, 1903.
"	"		1			Münchner med. Wochenschrift, 1903.
Freund	Mikuliz	9 weeks	1	5 months		Mitt. aus den Grenz. der Med. u. Chir., 1903
Greef	Bull		1	12 hours		ARCHIVES OF PEDIATRICS, 1903.
Gallant			1			ARCHIVES OF PEDIATRICS, 1903.
Schotten	Jackhs	35 days			1	Sammlung Klinische Vorträge, 1904.
"	Braun	45 "	1	20 "		Sammlung Klinische Vorträge, 1904.
Shaw	Elting	16 months			1	
			12		7	Mortality, 63 per cent.

SURGICAL CASES.

PYLORECTOMY.

Physician.	Surgeon.	Age.	Died	Time After Operation.	Recovered.	References.
Thompson	Stiles	8 weeks	1	10 hours		Brit. Med. Journal, 1902 Mortality, 100 per cent.

PYLOROPLASTY.

	Brissand	6 weeks			1	La Semaine Medicale, 1900.
Lange	Braun	9 "	1	20 hours		Münchner med. Wochenschrift, 1901.
Graanboom	MacGillav	4 "	1	6 weeks		Monatschrift für Kinderheile, 1902.
Cautley	Dent	8 "	1	3 months		British Journal for Diseases of Children, 1903
"	"	6 "			1	British Journal for Diseases of Children, 1903
"	"	7 "			1	British Journal for Diseases of Children, 1903
"	"	5 "			1	British Journal for Diseases of Children, 1903
Guthrie	Murray				1	London Society for the Study of Diseases in Children, 1902.
			3		5	Mortality, 38 per cent.

DIVULSION OF THE PYLORUS (LORETA'S OPERATION).

Ritchie	Nicoll	6 weeks			1	Brit. Med. Journal, 1900
	Schmidt	8 "			1	Arch. f. Klin. Chir. Langenbach, 1901.
Still	Burghardt	8 "			1	Brit. Med. Journal, 1901
Thomson	Stiles	3½ "	1	48 hours		" " " 1902
"	"	4 "			1	" " " "
"	"	5 "	1	2 weeks		" " " "
"	"	11 "			1	" " " "
"	"	5 "	1	36 hours		" " " "
"	"	3 "	1	4 days		" " " "
Mackay	Mackay	6 "	1	12 hours		Intercolonial Medical Journal, 1903.
Lendon	Wigg	6 "	1			Australia Medical Gazette, 1902.
			6		5	Mortality, 55 per cent.

NOTE.—Küster opened the abdominal cavity in one of Schotten's cases, but did not perform a gastroenterostomy as intended, on account of the feeble condition of the child.

SOME CONSIDERATIONS REGARDING SUBSTITUTE FEEDING DURING THE FIRST YEAR.

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The scope of a paper of this kind is too limited to allow of an exhaustive and detailed consideration of the possibilities and the resulting requirements of substitute feeding. What only can be done is to give briefly some idea of the various parts of the subject which our experience has shown to be important as a basis for rational treatment. There are certain essential principles which govern the modern method of feeding in the first year. It should be understood, however, that this substitute feeding even when dealing with the ordinary cases and not with the more difficult ones is not a simple problem.

We should remember that in a large proportion of cases the question of substitute feeding is not one of choice but of necessity, and that it is not a question of the relative value of natural and artificial methods. The great mass of cases of substitute feeding represents failures of breast feeding, either because the mother will not or can not nurse her infant. In like manner, whatever the cause, the great majority of these failures of breast feeding could have been avoided and the infant fed upon a substitute food successfully, provided that the modern methods, especially including the principles of percentage feeding, could have been instituted early enough and intelligently followed.

I. HUMAN MILK AS A GUIDE IN SUBSTITUTE FEEDING.

We have in the last decade obtained considerable additional knowledge regarding human milk, and we still acknowledge that human milk feeding is best for the human infant, if such milk is obtained from a mother whose nervous system and general characteristics render her fit to nurse her infant. Human milk, however, has many limitations arising from the fact that the mammary gland is so sensitive that many causes alter its products and thus contraindicate the employment of these products as a food.

The question which must continually arise is what materials and what methods have been found by the latest practical and scientific investigations to be best adapted to the infant when good breast milk cannot be obtained. Allowing that human milk is the best food for infants in the first year of life the subject of substitute feeding must be dealt with as any other life saving question. It is evident, however, that we must first understand what it is that makes human milk the best food for infants and from our study of this subject we have arrived at certain conclusions which, although not entirely satisfactory, may be distinctly and concisely stated before taking up the question of materials and methods for substitute feeding.

In leading up to the subject of substitute feeding we must deal with the subject of human milk only in a very general way. The essential principles to be recognized and emphasized are as follows:—Human milk is practically sterile as it enters the infant's mouth and the temperature is 98° to 100° F., it is amphoteric or slightly alkaline in reaction. Although it is well known that various salts differing in their proportions from those in human milk are held in solution, modern investigations have not been carried far enough for us to obtain any practical clinical advantage in attempting to modify these salts when the milk of another mammal is used as a substitute food. The three broad representatives of food stuffs, fats, sugar and proteids, which occur in human milk, are the ones which we must practically deal with when making a substitute food. These three essential elements forming the material to be used for the nutrition of the infant do not always occur in the same percentages in milk from healthy women. Human milk, therefore, varies within certain limits and may, therefore, be considered a variable food, a point of great importance and one which must be emphasized in substitute feeding. Thus good human milk may show a percentage of fat varying from 3 to 4, sugar from 6 to 7 and proteids from 1 to 2. Practical observations, however, of breast-fed infants show that in individual cases the limits in the percentages just given are not absolutely necessary for nutrition and do not mean that a somewhat higher percentage of fat than 4 must necessarily be unfit for the infant, nor that a decidedly higher percentage of the proteids than 2 must necessarily be undigestible, nor that the infant cannot thrive on less than 3 per cent. of fat or 6 per cent. of sugar. What it does mean, is that certain percentages and combinations

of percentages of these essential elements, fat, sugar and proteids, must be adapted to the digestion and nutrition of the especial infant, and that no one set of percentages will fulfil these conditions whether they occur in human milk or in a substitute food. This simply means that a successful substitute feeding so far as percentages are concerned is essentially an adaptation of special percentages to the individual infant.

Certain additional facts in regard to the fat, sugar and proteids of human milk should be noted. The fats are evidently provided not only for the purpose of nutrition but as a means of maintaining bodily heat. This function of the fat cannot with impunity be trifled with, for it is essential for the active metabolism, the importance of which we are forced to recognize when we attempt to provide a substitute food. The proper amount of fat in the food is probably of great aid in the regulation of the fecal discharges and for this purpose varies in the individual infant according to its especial idiosyncrasy. It is also presumable that, for the proper digestion of the proteids, the fat in the individual case should be in proportion to the proteids. This, however, is practically a part of the problem which has not yet to any extent been solved. We have noted that breast-fed infants thrive best when the percentage of fat is nearest to the maximum and that when this percentage is high for the individual infant there will be disturbance of the digestion shown by a tendency to diarrhea or vomiting, while when such percentage is less than demanded by the individual infant the nutrition suffers and there is a tendency to constipation.

There are many cases, especially in premature babies and in babies with weak fat digestions, in which greater variations than normally occur in human milk are called for and where very low percentages of fat may be necessary.

The form of sugar which nature has found to be practical in the early months of life, and therefore provides for in the milk, not only for human infants but for all mammals who suckle their young, is milk sugar or lactose. It is well to remember that although this sugar is more digestible than fat that it does not have so much heat producing power in a given weight, its potential energy to the fat being only 1 to 2.4. It is also important to remember that it is milk sugar which in normal digestion is acted upon by the bacillus lactis aerogenes to produce an organic acid which drives out the more noxious forms of bacteria, which by

their presence interfere with normal digestion. It is of some significance in the preparation of a substitute food that the nitrogenous constituents of human milk are represented by a number of distinct elements, yet practically our knowledge of these elements, so far as their use in substitute feeding is concerned, is limited in a general way to caseinogen and to the proteids of whey. It should be remembered in regard to these proteid elements that a supposed characteristic of human milk is that the whey proteids are in larger proportion than is the caseinogen and according to König this proportion is about 2 to 1. Although all chemists do not agree upon this percentage relation between the whey proteids and the caseinogen, and although there are many questions as to the whey proteids of milk, which are still undetermined, yet there is not much doubt but that these relative percentages in human milk differ much from those which occur in animal milks and therefore are an important consideration in preparing a substitute food.

Assuming good human milk as a standard we see by comparing its percentage ingredients with those of the milk of other mammals that there is no other milk which approaches its percentages sufficiently closely to permit of its being given in a purely undiluted form. We can also conclude that it is not an intolerance for milk as a whole, which causes so much difficulty in a substitute food, but that it is an incapacity of the individual infant to digest certain ingredients of milk. The fact which has been noticed by those who have regulated their substitute foods on a percentage basis is that there is a variable ability of the infant to digest fats, sugar and proteids, giving rise to what may be spoken of as fat indigestion, sugar indigestion and proteid indigestion. It is obvious, therefore, that the best system for obtaining a substitute food must provide not only for different percentages of fat, sugar and proteids, but also that these variable percentages can be obtained in any desired combination.

II. COW'S MILK AS A SUBSTITUTE FOR HUMAN MILK.

It is at once seen by referring to such mammals as the goat, ass and cow, mammals whose milk is most readily obtained in most countries, that a simple dilution of these milks with water, although in fact a modification of milk, in no way makes any of these milks resemble human milk sufficiently to allow of such simple dilution. Practically it is the cow whose milk we in most

cases must use and it is, therefore, well to remember in our substitute feeding one or two points in regard to the milk of the different breeds of cows. Although the percentages in the milk of different breeds vary as they do in the individual animal, yet this is not of great importance. The fact is that the milk of whatever mammal is used or whatever breed of that mammal, whether in combination with artificial foods or not, modification is the essential principle and that it is about as easy to modify one set of percentages as another. The essential principle is that we should modify the different elements and percentages in milks as exactly as possible in order that we shall be able to adapt the combination of percentages to the greatly varying digestive capacities of the individual infant. In substitute feeding modification in one way or another is always employed and it is merely a question of proper modification. This does not mean that precise and unvarying percentages are necessary in every, or even in the majority of cases of infants, but that we do need exactness in our percentage modification of milk to determine intelligently what variations can be safely allowed in the especial case. There is no doubt from my experience and from that of many others that in certain cases even slight variations in the percentages of a substitute food produce effects either good or bad. It is also an essential principle which I think has been recognized by every one that while we emphasize strongly the possibility and desireability of exact percentage modification in substitute feeding, yet the other cardinal principles are just as important, namely:—an absolutely clean and a bacteriologically safe milk-supply. This means that pure materials must be obtained first and the modification of these materials second. The modification of impure milk must necessarily be disastrous to the infant and a bad modification of pure milk can be almost equally disastrous.

We are justified in saying that routine mixtures cannot be relied upon any more than can indiscriminate breast feeding. We can also state that, judging from the clinical observations of a large number of breast-fed infants, these observations suggest and in fact emphasize the principle that in the management of infant feeding we must recognize two important conditions. One of these is the digestion of the infant, and the other is its nutrition. These two requirements are essential for a successful lactation whether breast or substitute. Milk may be easily digested but not nutritious, or it may be highly nutritious but difficult to digest so

that it is the equilibrium of these two conditions which produces a perfect infantile development. Supposing that the cow is the mammal from whose milk we decide to make our substitute food, a few points concerning cows are of practical importance besides the mere differences in the percentages of the different elements. The higher grades of cattle have been found not to provide the best milk for a substitute food. The fats for instance in the milk of the Jerseys and Guernseys differ very considerably from that of the other breeds and also from human milk, and it is preferable to use the milk of such cows as Holstein, Ayrshire, Durham and Devon in whom the fats appear to be more digestible and more in accordance with those of human milk. It is to be noted that the milk of the Holstein has a much larger proportion of the stable fats such as olein, stearin and margarin than does that of the Jersey which contains a much larger proportion of the volatile glycerides. These volatile glycerides do not exist in the mamma, but are formed in the milk soon after milking and this change takes place more quickly and notably in the milk of the Jersey and Guernsey cows. In comparing also the milks of Holstein and Jersey cows we find that in the modification of the former, the emulsion is more easily restored, is more permanent and is not so likely to produce oil-drops on the surface of the milk when subjected to the influence of heat and handling. The latter fact is, of course, of considerable importance when we are dealing with the rather difficult question of the transportation of modified milk.

It is important in a general way to know what effect heat has upon milk. 155°F. is sufficiently high to kill practically all pathological germs found in milk without coagulating the lactalbumin or producing any change which can be detected by the taste or appearance of the milk. This temperature is also sufficient to destroy the rennin enzyme which is used in obtaining the whey proteids and yet not high enough to coagulate the whey. Care must be taken when higher temperature such as 212°F., and sometimes even when lower temperatures are used not to combine a high percentage of sugar with lime water higher than 10 per cent., as under these circumstances the mixture is liable to become brown, seemingly from the action of the heat, the milk sugar, and lime water in producing a caramel. It is possible also that certain organized principles at a heat of about 177° F. undergo a change the nature of which is not very well known, but the evidence of which is found in certain chemical reactions. It is, therefore,

better until more is practically known of the effects of heat on the milk above the required necessary 100° F., when given to the baby, not to use a temperature over 155°F., excepting in cases of transportation in hot weather, and in fact when we are sure of a clean milk-supply not to pasteurize at all.

If we have decided, as I am taking it for granted we have, to consider cow's milk as the substitute for human milk, it is well to know what the composition of the milk is in order that we can modify more exactly. We presuppose that the milk is not Jersey or Guernsey, but from a common mixed breed of cows preferably Holstein. The average of a large number of milks of this kind shows that the fat is 4.00, milk sugar 4.75 and total proteids 3.50. We must also understand that while in our standard human milk the whey proteids are probably larger proportionately to the caseinogen, in cow's milk it is the reverse, the caseinogen making up four-fifths ($\frac{4}{5}$) of the total proteids. This, of course, must be taken into consideration, especially when we are arranging for a substitute food for young infants or for infants with a weak proteid digestion which usually means weak caseinogen digestion.

III. PERCENTAGE MODIFICATION OF COW'S MILK.

In making up a substitute food from milk there are certain materials which are found necessary in order to carry out the various percentage combinations; these materials are:—(1) Creams of different but definite fat percentages obtained either by gravity or centrifugal separation. (2) Fat free milk which is used to increase the percentage of the proteids in the food beyond that contributed by the cream. (3) Milk sugar to raise the percentage of the sugar above the amount contributed by the cream and fat free milk. (4) Lime water to increase the alkalinity. (5) Boiled water as a diluent. (6) Cereal water and jellies of barley or oatmeal to be used in definite percentages.

It is not necessary to speak of the details of obtaining these materials. We each have our own definite ideas as to the use of centrifugal or gravity cream, but it is well to remember that the fresh cream obtained by the centrifugal process is often of a good deal of value in the success of the treatment. What is most important in connection with the percentages, however, is that it should be understood that different creams are needed to obtain different combinations of the fat and proteid percentages. A few instances of this will suffice to illustrate the principle involved.

For instance, the lowest total proteid that can be obtained from a 10 per cent. cream, when 4 per cent. of fat is needed in a substitute food, is 1.34, while with a 16 per cent. cream a proteid as low as 0.80 can be obtained. We must, therefore, remember that there are limitations in the percentage of the proteids dependent upon the use of creams of different strength. Other examples of the principle are, for instance, that a 3.50 fat, 6.00 sugar and .50 proteid cannot be obtained with a 10, 12, or 16 per cent. cream, but only with a 20 per cent. cream. Again, a 2.50 fat, 5.00 sugar, .50 proteid cannot be obtained with a 10 or 12 per cent. cream, but can with a 16 or 20 per cent. cream. Again, a 4.00 fat, 7.00 sugar and 1.00 proteid, a combination which is very generally used, cannot be obtained with either a 10 or 12 per cent. cream, but can be had with a 16 per cent. or 20 per cent. cream. There are many examples of this kind. We must also know what combinations may be obtained when we wish to use either total proteids, such as are obtained in plain cream mixtures; or split proteids, such as are obtained in the whey-cream mixtures. The possibilities of such combinations are seen in Table A.

TABLE A.

Prescriptions for modified milk may call for percentages of fats, sugar and proteids, in variations of 0.25 per cent. Any percentage of fats from 0.00 to 4.00 may be obtained in combination with any percentage of sugar from 4.00 to 7.00, proteids from 0.25 to 3.50

WHEY CREAM MIXTURES.

Any percentage of fats and sugars as given above may be obtained with the following combinations of whey, proteids and caseinogen.

WHEY PROTEIDS, PER CENT.	CASEINOGEN, PER CENT.
.25	.25
.50	.25
.75	.25
.90	.25
.90	.50
.90	.75
.75	.50
.90	.60
.90	1.00
.75	1.25

NOTE:—If an alkalinity above 5 per cent. is desired in whey cream mixtures over 3 per cent. fat, the whey proteids cannot practically exceed 0.80 per cent.

I shall not say anything more about the materials for substitute feeding, having mentioned the principal precautions to be taken. The remainder of what I have to say about substitute feeding will presuppose a modification of cow's milk always as fresh, as clean and as nearly sterile as is possible in the individual case which is being managed.

IV. USE OF CEREALS IN SUBSTITUTE FEEDING.

The question of the use of cereals during the first year of life is an interesting one and one which has for many years been either advocated or opposed by those who have studied substitute feeding. Dr. Henry Shaw, of Albany, has contributed lately (January, 1904), some excellent work on starch digestion in infancy, and his results confirm what has been known for some time that the saliva of even very young infants contains a diastatic enzyme capable of converting small amounts of starch into maltose. This amylolytic function of the infant gradually increases as the infant grows older and seems to be well developed by the end of the first year. The small amount of starch which is digested in the early months is probably of little value from a nutritive point of view, the principal function of cereals at this early period of life being as an attenuant in modifying the caseinogen of cow's milk. It seems that as the standard human milk presents fewer failures in feeding than any other food and contains no starch it would be well to avoid using cereals, if possible. Since by the modification of the caseinogen to 0.25 by using the split proteid, and at the same time using a high whey proteid, the total proteid can be kept over 1.00, it would seem that this latter method of treating the proteids is more rational and more in accordance with natural physiological laws. It is possible, also, that merely because the diastatic power starts at birth, that it is not necessarily intended to be used at that time, as it is still an undeveloped function and is liable to be over taxed if used too early. The use of cereals should not only be instituted according to the age but also according to the diastatic power of the individual infant since there is no doubt that some infants seem to need and thrive on cereals much earlier than do others, just as some infants can digest and thrive on much larger percentages of the caseinogen of the cow's milk at a much earlier period than can others. What, however, is of special importance in this question of starch is that the physician should not only decide in the given case, whether to give a cereal but also

should know what strength of cereal he is giving so that the especial infant may receive the percentage of starch which is best for it as it does the percentage of fat, sugar and proteids. This I have long advocated and Dr. Maynard Ladd is at present working out a system at the laboratory by which we can order any percentage of starch which we wish in combination with the other percentages of fat, sugar and proteids. While I have not found the use of cereal solutions necessary or advisable in the early months, yet, later, when they become of value in certain cases and also in the final weaning from modified milk to whole milk, an exact system will, I believe, be a most valuable adjunct to our feeding.

Cereal solutions can be added in place of the boiled water in any combination without altering the percentage of the fat, sugar and proteids, and the percentage of the starch thus added may be accurately determined, if the percentage strength of the cereal solution is known. For instance, if 14 ounces of a 1 per cent. solution of barley water is added in place of the boiled water to a 20 ounce mixture, the percentage of the starch in the mixture is $\frac{14 \times 1}{20} = 0.70$.

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V. THEORY AND PRACTICE OF PERCENTAGE FEEDING.

Next of importance to the purity of the milk supply in obtaining a successful result in percentage feeding is that the proper amount, according to the age and weight of the especial infant, should be given and that the food should be given at proper intervals according to the individual infant and not necessarily according to the age. Too large or too small an amount of a proper food and to frequent or too prolonged intervals of feeding are responsible for many failures in the feeding problem. There is no question but that certain infants under certain circumstances do not require as much as the average infant of the same age, and it is often necessary to either change from shorter to longer intervals, or from longer to shorter ones than is usually found to be best in the average cases. From what has already been said, I think that the indications for the use of the various percentage combinations are quite evident.

Our knowledge of the physiology and chemistry of human milk as compared with cow's milk is, we must acknowledge, imperfect, and we must not, therefore, expect that an infant in the early weeks and months of life can in all cases take a combination of percentages which corresponds to human milk but which is derived

from cow's milk. We, therefore, in starting a new-born infant upon a modified milk take the precaution of beginning with percentages much below the standard average percentage of human milk and usually lower than the especial infant would probably take. This rule is prophylactic, for it is a much simpler matter, having once determined what the especial infant can digest, to adapt the food by gradually increasing the percentages until the nutrition is good, than to give a highly nutritious food in the beginning and thus run the risk of digestive disturbance.

The most frequent mistake in substitute feeding is to give a milk of too high percentages. It is most important, whether we are starting with a premature infant or with an infant at term, to begin with low modifications and then, advancing one element after another, to approach gradually the goal which we hope to attain by the eleventh or twelfth month, namely, unmodified cow's milk. This means as a rule that the percentage of the fat and the sugar should in most cases be rapidly advanced to 4.00 and 7.00, while the proteids usually have to be advanced more slowly and with greater caution. The whey proteid as a rule can be advanced more rapidly than the caseinogen, but we must always keep in mind that while the whey proteids are more digestible and, following the example of their proportion in human milk, can for some time be kept proportionately greater than the caseinogen, that the caseinogen probably is the more nutritious, and it is the percentage of the caseinogen which, as the months of the first year go by, should by the end of the year be increased both totally and proportionately so as to exceed the percentage of the whey proteids. We should also remember that while from the beginning to the end of the first year the fat when once a 4 per cent. has been reached remains the same, yet the sugar should be gradually diminished from 7.00 to 5.00 per cent. The rapidity with which these changes can be made varies according to the idiosyncrasies of the infant.

I have endeavored to show in Table B how the percentages of the fat, sugar and total proteids, or whey proteids and caseinogen, should be advanced week after week, accompanied by a corresponding increase in the amount given at each feeding and in the intervals between the feedings. The number of the feedings in the twenty-four hours is also shown in the last column. These figures I have found to be, however, only a general guide and in this sense are theoretical, although they have been deduced from actual cases and for quite a large majority of infants they will be found

to be fairly accurate. No table can show us how to feed in every case the special infant and I must reiterate that each infant should be studied as a problem in itself, and that we are merely aided in the solution of the problem by such tables as these and by keeping in mind the general principles spoken of earlier in this paper.

TABLE B.

THEORETICAL BASIS FOR FEEDING A HEALTHY INFANT.

Age	Fat	Sugar	Proteids	Proteids if split		Amount at each feeding in		Intervals between feedings in hours	Number of feedings in 24 hours
				Whey proteids	Caseinogen	ounces	c. c.		
Premature.	1.00	4.00	0.25	0.25	0.25	½–¾	4–25	1–1½	24–18
Premature.	1.50	4.50	0.25	0.50	0 25	"	"	"	"
At term.	2.00	5.00	0.50	0.50	0.25	1	30	2	10
End of 2d week	2.50	5.50	0.50	0.50	0.25	1½	45	2	10
" 3d "	3.00	6.00	0.75	0.75	0.25	2	60	2	9
" 4th "	3.50	6.50	1.00	0.75	0.50	2½	75	2	8
" 6th "	4.00	7.00	1.00	0.90	0.60	3	90	2½	8
" 8th "	4.00	7.00	1.25	0.90	0.75	3½	105	2½	7
" 12th "	4.00	7.00	1.50	0.90	1.00	4	120	2½	7
" 4 months	4.00	7.00	1.50	0.75	1.25	4½	135	2½	6
" 5 "	4.00	7.00	1.75	....	....	5½	165	3	6
" 6 "	4.00	7.00	2.00	....	....	6	180	3	6
" 8 "	4 00	7.00	2.50	....	....	7	210	3	6
" 9 "	4.00	7.00	3.00	....	....	8	240	3	6
" 10 "	4.00	6.00	3.00	...	....	8	240	3	6
" 11 "	4.00	5.00	3.00	....	....	10	300	3	5
" 12 "	4.00	4.75	3.50	....	....	10	300	3	5

The data given above are simply a guide to the rules for the feeding of the average healthy baby; they are only approximate and not intended to be followed in cases of difficult feeding without due consideration to the individual requirements.

Starting with a premature infant with a fat percentage of 1.00, sugar 4.00, whey proteids .25 and caseinogen .25 or, with an infant at term with a fat percentage of 2.00, sugar 5.00, whey proteids .50 and caseinogen .25, we should endeavor by the end of the third week, in the case of the infant born at term, to have attained a 3.00 fat, 6.00 sugar, .75 whey proteids and .25 caseinogen, and then, gradually increasing, at the end of the sixth week we can in most instances attain a 4.00 fat, 7.00 sugar, .90 whey proteids and .60 caseinogen. At the end of the fourth month a 4.00 fat, 7.00 sugar, .75 whey proteids and 1.25 caseinogen may be reached, while at the end of the fifth or sixth month in many cases it is

found that the infant thrives better if given a total proteid instead of the split proteid, even if smaller percentages of the total proteid are given, for in this way we use in larger proportions the more nutritious element, caseinogen.

In the first twenty-four or thirty-six hours of life in most cases, especially while the physician is waiting to see if the mother will be able to nurse her infant, a dram or two of a 5 per cent. lactose solution in boiled or distilled water is sufficient. After that a modified milk can be begun with at once. The rule is to begin with a low fat—1.50 to 2.00. The time for giving 4.00 fat varies greatly but usually is somewhere from the fourth to the eighth week. Higher than 4.00 fat I have not found it advisable to give except in a few cases. The 7.00 of sugar in many cases can be attained quite early in the first, second or third weeks, although usually it is reached, as shown in the chart, about the sixth week.

In regard to the proteids, it is often disastrous to give them too high to the infant in the beginning, and many physicians, I think, err in giving them so high as to cause disturbance of the digestion and of nutrition. There is, on the other hand, much danger in keeping the proteids too low and it is exceedingly important that the physician should follow his case carefully, and see that the changes from time to time are tried until he is sure that the nutrition of the infant is not suffering from a too low caseinogen percentage. The physician must discriminate nicely between the two extremes of too low and too high caseinogen percentages. The rule is to begin low, watch carefully, increase gradually and at once decrease if symptoms of indigestion appear.

The use of split proteids and the change to the total proteid is a matter of judgment in the individual case. In many cases of weak digestion or if the infant is sick, the split proteid is found to be of great value even in the latter months of the first year; here again, however, the idiosyncrasy of the infant is strongly shown and must be determined in the individual case. In the ninth, tenth or eleventh month, according to the individual infant, the percentage of the total proteids should, if possible, be raised to 3.00 and the sugar reduced to 6.00 and again to 5.00. When we finally arrive at the goal which we have been striving for, namely, a 4.00 per cent. fat, 4.50 or 4.75 sugar and 3.50 proteids the modification of the milk can be stopped and unmodified cow's milk be given.

Symptoms.—It is not in many cases by any means a simple

matter to decide which element of the food is at fault from the usual symptoms of gastroenteric disturbance. Sometimes it may be one element and sometimes another and yet the symptoms being very similar we cannot always determine which is responsible. It may be an over amount of one element, or an improper combination of elements which may result in a failure to gain in weight so that it is only by beginning at once in the more intractable cases with low percentages of the various elements and combining them in various ways that we can finally solve the problem.

Peptonization.—In certain cases, although in my experience rarely, peptonization, either partial or complete, of a modified milk may benefit the especial case. If, however, we diligently and intelligently carry out the essential principles underlying the adaptation of cow's milk to the infant and the more familiar and experienced we become with the principles of percentage feeding the less frequently will we resort to peptonization.

Accurate Weighing.—Probably the most important aid which we have in adapting a substitute food to the especial infant is the knowledge obtained by constant and accurate weighing. The variations in weight whether in gain or loss are so significant in connection with the question of the infant's nutrition that we are working as much in the dark when we do not have this aid to our treatment as when we are working with empirical formulæ instead of known percentages. A steady gain almost always indicates that we are working on the right principles, while an arrest or loss in weight indicates the opposite. A valuable guide as to the weight development of an infant is to be had by keeping a record of the weight index. The significance and convenience of this standard of comparison has been presented to you at this meeting by Dr. Ladd. The weight index expresses the ratio between the weight of any infant to the weight of the average normal infant of the same age. Expressed in per cent. it represents the weight development of an infant compared with the average healthy infant whose percentage development we may reckon as 100. The variations from the weight index at birth give a more accurate idea of the progress of an infant in its weight development than can be obtained by noticing the number of ounces gained or lost. For a means of comparison with other infants of different ages and stages of development, it is of obvious advantage to express the weight development in definite percentages.

Failures.—We should always remember that in a large number

of feeding cases failures are pretty sure to occur, but we must remember in judging of our results that these failures are not always due to the food. It is a mistake that is frequently made by the physician as well as by the laity always to attribute loss of weight and gastroenteric disturbance to a fault in the character of the food, while in reality they may occur from faulty metabolism and unknown causes of bacteriological infection of the intestinal tract, of inhalation poisoning, undetected malformation of the gastroenteric tract, unhygienic surroundings and many other causes.

Final Remarks.—Let me reiterate what I have said before: infants cannot be fed by rule of thumb. We can only lay down the general principles governing the milk-supply and subsequent modification of the milk and, finally, its adaptation to the individual infant. Each infant must be studied by itself. The most successful feeder of infants will be the man who is thoroughly acquainted with the many resources in our modern methods of percentage feeding and who can appreciate the individual needs of the especial infant.

DISTURBANCES OF DIGESTION IN INFANTS, RESULTING FROM THE USE OF TOO HIGH FAT PERCENTAGES.

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That serious disturbance of digestion in infants may be due to an excess of any of the milk constituents in the food employed, is a trite remark, and is the daily experience of all who see much of the results of infant feeding. The practitioners and the skilled nurses who direct the feeding of infants have come pretty generally to appreciate the symptoms which usually follow too high proteids, to understand what percentages of proteids an average infant may be expected to digest under given conditions, and how these percentages may be secured in the various milk formulas used. With the methods of home modifications of milk now in vogue, it is probably true that the proteid percentages of our formulas have been rather lower than higher than we have calculated, but often to the decided advantage of the patient.

But how is it with the fat? Personally, I have reached the conclusion that disturbances of digestion resulting from an excess of fat are quite as serious, if not quite so obvious, as those which follow the use of too high proteids, and that they need to be studied just as carefully. Even those who give special attention to infant feeding have been slow to learn the infant's capacity with respect to the digestion of fat, both in health and in the ever-varying conditions of disease. Others who have given the question less attention have fallen into error in one of two ways: In following the formulas of the books instead of using an ordinary milk, with a fat content of 4 per cent., they have substituted a rich Jersey milk containing from 5 to 5.5 per cent. fat. Thus unwittingly they have given from 5 to 7 per cent. fat when they had supposed they were giving a food containing 3.5 or 4 per cent. Or, they have made another mistake of intentionally increasing the fat, almost without limit, for the purpose of over-

coming that most troublesome symptom in artificial feeding—chronic constipation.

Errors like these are exceedingly common and their consequence may be very serious. It is the purpose of the present paper to record some of the results seen with such feeding, all of the cases cited having come under observation in private practice in the course of a few months.

CASE I.—*Over-feeding with high fat, rapid increase in weight and progress in development till eight months; then general convulsions, followed later by tetany, laryngismus stridulus, fatty liver* (?). *Recovery after three months' illness.*

In February, 1904, I was asked by a physician to see a baby of a medical friend whose feeding he had directed. The child had been considered a magnificent specimen of physical development, and a great triumph of the feeding art, until one week before my visit, at which time his weight, at eight months old, was twenty-one pounds. In the previous three-and-a-half months he had gained ten pounds. The child had bright rosy cheeks; was strong in his muscles, already pulling himself up by the side of his crib and bearing his weight on his feet; and was for his age well advanced mentally. He seldom vomited; had a good appetite, and was taking, until a few weeks before I saw him, seven feedings of 6 ounces each, but lately only six feedings, of a mixture made up as follows:

The upper 16 ounces were taken from each of 2 quarts of a rich Jersey milk (Briarcliff Dairy); 24 ounces of this top milk were used in a 40-ounce mixture. Examination of the top milk used showed it to contain 10 per cent. fat, so that the baby was receiving in his food not less than 6 per cent. fat. With this food the bowels usually moved twice a day; the passages being smooth, large and yellow. An occasional restless night and some transient urticaria were the only clouds in the otherwise clear sky until February 16th, when a stool not well digested was reported, with loss of appetite. These were not enough to make necessary any change in food.

The following morning, one hour after feeding, the child had a severe general convulsion which lasted from six to eight minutes, followed by a second one three hours later. The stomach was promptly washed out and all food stopped; calomel given and later castor-oil; then bromide of soda. Five loose stools followed this treatment, none of them especially foul. There was no

rise of temperature and no signs of serious illness. The following day, as no unfavorable symptoms had occurred, the previous food was begun, but much diluted, the proportions being one part of the food to five parts of water. The strength of the food was gradually increased, so that at the end of four days the baby was taking 5 ounces of his original food diluted with only one ounce of water. During the next night some severe pain occurred, ascribed to colic; the next morning the temperature was 104.5° F. I saw him first early in the afternoon. He was, as he had been described, a splendid looking child. The fat lay in great folds over his body and extremities. Fontanel large, but no other evidences of rickets; no teeth. Liver much enlarged, the lower border being nearly two inches below the costal margin. Hands and feet in typical position of tetany, considerable muscular rigidity, movements painful; temperature 103.5° F.

A second clearing out of the intestinal tract was advised, and the first stool passed was examined chemically and found to consist of almost pure fat. All food was omitted for one day, all milk for a week, and then very low fat percentages given. The tetany gradually passed away, but was followed three weeks later by laryngismus stridulus which continued for nearly three weeks. There was much trouble with the digestion for the next two months and the child lost 5 pounds in weight.

When last seen, three and a half months after his severe attack, he was digesting well and thriving, but his weight was only 19 pounds.

Several points of interest in this case deserve attention; (1) the long period during which the child bore the high percentage of fat without any apparent disturbance; (2) the severity of the nervous symptoms, the suddenness with which they came on and their long duration; (3) the prolonged disturbance of the digestion and general nutrition; (4) the large liver (*paté de foie gras?*) and stools consisting of almost pure fat.

CASE II.—*Prolonged feeding with high fat, notwithstanding which constipation and the development of moderate rickets, followed by acute disturbances of digestion with repeated convulsions.*

This patient was also seen in consultation within a few days of Case I, and the symptoms presented many points of similarity. The milk used was from the same dairy; it was prepared after almost the same formula, except that the percentage of fat in the

food as given was only 5 per cent. The patient was the first child of healthy parents, nursed for three months, had suffered somewhat from facial eczema at six months, after which barley water had been substituted for plain water as a diluent. For the past month the child had been taking the following formula:

Top milk.....	19 ounces.
Barley water.....	21 ounces.
Sugar of milk.....	4 even tablespoonfuls.
Milk of magnesia....	1 teaspoonful.

The top milk was the upper 16 ounces from 1 quart of rich Jersey milk and had been estimated by the physician to contain 7 per cent. fat. The child was taking 6 ounces every three hours. With the addition of the magnesia the bowels moved regularly, but the stools were pale yellow. The formula begun after weaning had been a similar one with a little lower proportion of milk. The baby was gaining from 6 to 8 ounces a week and his weight at the time of my visit, when he was eight months old, was 18¾ pounds.

There had been no vomiting, the child had a good appetite, slept well, and was considered to be in the best of health.

One week before my examination, without any change in the food or other assignable cause, the child had a slight convulsion in the evening. The following morning a second and more severe one. The temperature was elevated up to 103° F., and the child gave the appearance of being seriously ill. Food was stopped, the bowels cleared with calomel followed by magnesia, and the temperature quickly fell to normal. The child improved rapidly, and he was put back on his previous food, at first diluted, but strengthened daily until at the end of three or four days it was two-thirds the original strength. One week after the first convulsions, he had a third, and later in the day two more. I saw him in the evening. He presented all the appearances of a splendid, healthy, well-developed child with bright red cheeks, plenty of fat, and muscles well developed. The head was well shaped, but craniotabes was present; no teeth. Physical examination otherwise negative. All food was stopped for twenty-four hours and a second clearing with calomel ordered. All milk was stopped for a week and then a mixture containing a low fat substituted. Three months later I saw the child again. He had one convulsion six weeks after the first group, apparently from a slight at-

tack of indigestion. At the last visit he was in excellent condition, still no teeth at eleven months, with moderate head sweats, but general condition otherwise excellent. The craniotabes had disappeared.

It was interesting to note the development of rickets and the presence of constipation, in spite of the excessive amount of fat which his food had contained ever since he had been weaned at three months.

CASE III.—*Over-feeding with high fat—convulsions.*

This was an office patient six months old, the second child of healthy parents, whose weight at birth was reported as 8¾ pounds. The mother had nursed for a few weeks; then discontinued on account of illness. The child had been fed upon a milk modified at home; the proportions were stated to be as follows:

Milk	12	ounces.
Cream	4½	"
Lime water	1½	"
Water	14	"
Total	32	"

The child was given 5½ ounces every three hours. The cream used was examined and found to contain 35 per cent. fat. The proportion of fat in the food was thus about 6.75 per cent. Upon this mixture the child had gained very rapidly in weight, had had from three to four yellowish stools daily, occasionally containing masses called curds, and its progress had been considered most satisfactory until the age of four months. There had been some vomiting, but this had never been very troublesome. At four months old a severe general convulsion occurred lasting twenty minutes, a second convulsion two hours later. The food was reduced to a mixture of equal parts of milk and water, which was continued for a few days; but as the child did not gain as rapidly as before, the mother on her own responsibility had returned to the original formula. The father, a dentist, being convinced that the convulsions were from dentition, ascribed great benefit to gum-lancing, which was practised. After the first convulsion the general condition was not so good, slight symptoms of indigestion were present—regurgitation of food many times a day, much flatulence and stools usually yellow, but occasionally green.

Examination showed a moderately fat infant, pale, flabby, without signs of rickets.

The cream was omitted from the formula and milk substituted reducing the proportion of fat to about 2 per cent. The child made satisfactory progress from this time, had no further convulsions and its symptoms of indigestion rapidly disappeared.

CASE IV.—*Habitual vomiting aggravated by high fat; serious gastric catarrh produced; finally cured by stomach washing.*

This was an infant ten months old, also seen in consultation. The chief symptoms in this case were continued wasting with persistent vomiting. The vomiting had begun almost from birth and had been a most troublesome symptom up to the time of my visit. A great variety of infant foods and milk formulas had been employed, none with any very great success. Up to the eighth month the food had been for the greater part of the time barley water and condensed milk. For the past two months the child had been in the care of a physician who is enthusiastic in the feeding of high fat. The formula used had been:

Top milk	17	ounces.
Water	13	"
Lime water	2	"
Total	32	"

The child was given four feedings, 7 ounces each; the interval between feedings, four hours. Occasionally an extra feeding was given during the night. The top milk was the upper 17 ounces from 1 quart of the Briarcliff milk. According to two analyses of similar samples, the food contained as given about 5.30 per cent. fat. During the two months that the child had been on this diet, his weight had fallen from 9 pounds 8 ounces to 8 pounds 4 ounces. His vomiting had not improved; in fact, had grown steadily worse. Now not only was food rejected, but frequently mucus in large amounts at some time almost every day. The child had never had any acute disturbance of digestion; the bowels were constipated.

Examination showed a typical marasmus child. Head 16 inches, chest 14¼ inches, length 23½ and weight 8 pounds 4 ounces; at ten months his weight being only 2¾ pounds more than at birth. After one or two minor changes the child was put upon the following:

Plain milk	18	ounces.
Water	7	"
Lime water	5	"

This was peptonized. He was given 5 ounces every four hours; five feedings daily. Stomach washing was begun, at first daily and then every other day. Large amounts of mucus, often as much as two ounces at a time were removed. With the stomach washing the mucus rapidly disappeared and the quantity and strength of the food were increased. The stomach washing was continued at longer intervals for a period of nearly two months, during which time the weight steadily increased. At the end of four months he had gained 6¼ pounds. The progress from this time was rapid and uneventful, and one year afterward the child was reported by the attending physician to be fat, rosy and in the best of health.

The condition had been in this case persistent gastric indigestion with excessive fermentation, continuing until a gastric catarrh of serious proportions had developed. The high fat mixture which had been ordered had greatly aggravated this condition, and the state of the child at my first visit was really an alarming one.

CASE V.—*Feeding with high fat; eczema, habitual constipation, and finally, habitual vomiting.*

This case is an illustration of the very rapid gain in weight and subsequent development of serious digestive disturbance from very high fat. The child at birth was very large and robust, his weight being 11½ pounds. He had increased rapidly and at eight weeks old his weight was 15 pounds; at three months it was 17 pounds 10 ounces. The weekly gain during the greater part of the time was from 12 to 15 ounces. The formula used had been:

Top milk	24	ounces.
Water	22	"
Lime water	2	"
Mellin's food	3	tablespoonfuls.

The Mellin's food had been added to overcome the chronic constipation. The top milk consisted of 12 ounces removed from each 2 quarts of the Briarcliff milk, the same dairy as that which supplied the milk in three of the preceding cases. The proportion of fat in the food given was approximately 7 per cent. The mother

reported that the child had always been constipated, and that he had had no natural movement for three weeks, enemas or glycerin suppositories being usually required to induce one. The stools passed were large, dry, and a light yellow color. There had been a good deal of straining and, finally, a rectal prolapse had developed. The vomiting at first had been slight, but recently had greatly increased until it had become very troublesome and was repeated several times each day. He had an excellent appetite and seemed never quite satisfied with the food given. A troublesome eczema of the face had appeared about the third month.

It was interesting in this case to see that the constipation greatly improved when the fat in the food was reduced to 4 per cent.; but it was not entirely overcome until the fat was reduced to 3 per cent. The symptoms of indigestion and eczema continued to be troublesome for the next two or three months, but no serious symptoms occurred, and the progress on the whole was satisfactory.

It would be easy from my records of private patients to multiply almost indefinitely examples of acute and chronic disturbances of digestion in infants from the use of too high fat. Those cited, however, illustrate the most frequent and the most serious types. Often the very rapid and unusual gain in weight goes on steadily until the acute upset comes. In other cases the results of such feeding are shown in a gradual loss of appetite, sometimes so complete that for weeks a child will hardly take one-third of his former food. The increase in weight now ceases and there may even be a steady but a slow loss. In such cases the infant seems to take into his own hands the matter of cutting down his food and thus prevents serious acute disturbances of digestion.

That gastric disturbances—habitual vomiting or regurgitation of food, continued fermentation and finally the production of a gastric catarrh—may follow the use of very high fat has often been emphasized; but it is not so generally appreciated that disturbances of intestinal digestion may also occur, and that even chronic constipation may be greatly aggravated by such feeding. The hard, dry, gray stools passed under these conditions frequently consist almost entirely of undigested fat. Steadily raising the proportion of fat because of the constipation is the plan often followed, and this makes matters worse.

Severe nervous symptoms, such as occurred in several of the cases, have not in my experience been very frequently seen from

this cause, and I do not feel convinced that the form of auto-intoxication present was entirely due to the fats, although this is possible. The symptoms were not very different from those which accompany other forms of acute digestive disturbance.

There are some practical conclusions to be drawn from these cases. It is evident that the errors into which both physicians and patients have usually fallen have resulted from their fondness for rich Jersey milk and cream. Although there is pretty general agreement among those who have given special study to the question of infant feeding that milk from other herds, containing from 4 to 4½ per cent. fat, is better, still a great majority of the profession and the public will turn from such milk as being thin and blue to the rich yellow product of the Jersey or Alderney herd. I believe that one of the chief causes of failure in feeding from the milk laboratory has been the use of too high fat, it being so easy for a physician under these circumstances to increase the fat percentage, particularly if his mind has been filled with the idea that thereby chronic constipation is likely to be overcome.

Mistakes like those above described may be avoided if the physician knows approximately the fat content of the milk, cream or top milk which he is using. Too often he falls into error by disregarding this entirely and treating all specimens of milk and cream alike, using them in the formulas which he may have obtained from some book. More definite results may be obtained in several ways. In the first place, the physician may obtain from the milk laboratory a cream or milk of a definite fat percentage and thus be able to calculate closely the amount of fat which his patient is taking in the formula given. Or, he may himself examine the milk, top milk or cream, which his patients are using. His advice is generally asked in the choice of the milk supply and if he habitually uses milk from the same dairy he can be fairly certain of his results. Such examinations with the Babcock tester, or some of its modifications, can be made in a few minutes, or a specimen may be sent to a laboratory and the fat determination had for one dollar.

In order to interpret correctly the result of these milk examinations he must learn to think in percentages. In no other way can one form any idea of how much fat he is giving. Although a little difficult at first, it soon becomes easy. To a student of medicine there does not seem to be very much difference between a temperature of 103° and one of 106°, yet how different is the

meaning they convey to the practitioner. In the same way milk percentages at first signify little to one unaccustomed to using them, and where the increase is made by percentages at the milk laboratory, often the most injurious combinations are ordered. A friend told me of a physician who was feeding his own child on milk from the laboratory. The formula used was one containing 3 per cent. fat. As the child was not gaining very well, he decided that an increase was needed and ordered milk containing 6 per cent. fat. He then abused the laboratory because his baby's digestion had been upset by the milk.

As to how much fat may be wisely given to infants in health, opinions may perhaps differ somewhat. I am not sure that any arbitrary standard can be fixed. Children differ very much in their capacity to digest fats as in other respects; but there is a limit with each child beyond which we may not safely go. If the percentage is raised very much above this for any length of time disturbances are almost certain to follow. Personally, I have never seen any advantage, but often much harm, from raising the fat above 4 per cent., and in my own practice I have fixed upon this as about the limit for the average child.

The physician who assumes to direct the feeding of infants must have some notion of what is proper, must know what he is giving and must appreciate the difference between giving a food which contains 3 or 4 per cent. fat and one containing 6 or 7 per cent. Although the bad results of the higher percentages may not be at once apparent, they are almost certain to come later. Furthermore, simply to raise the percentage of fat seldom relieves chronic constipation; and, finally, whenever there are marked symptoms of either gastric or intestinal indigestion, the fat should be reduced much below the normal 3 or 4 per cent.

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DISCUSSION.

DR. NORTHRUP.—I am particularly interested in Dr. Holt's paper and would like to call attention to some experiments made at the Presbyterian Hospital, New York, with rickety children. Believing that they should have cod-liver oil, I decided to save the cod-liver oil bill by super-fatting the milk. As a result the children became constipated with hard, dry, crumbling feces and vomited, showing some irritation of the stomach. I am fully con-

vinced of the undesirability of using above a 4 per cent. fat. I am pleased with the paper because it coincides with my own views on this matter regarding the amount of fat. Sometimes, however, there are many difficulties in overcoming the prejudices of grandmothers in regard to proper milk for the babies. The key to the situation in infant feeding I got from Dr. Rotch: Keep the formulas "level"; not giving way to violent changes and disturbances of proportions.

DR. KERLEY.—With regard to the high fat feeding, in the New York Infant Asylum, I have never been able to use a higher fat than 2 per cent. to children under six months of age. I find they are less susceptible to intestinal derangements and thrive better in every respect upon a low fat with a moderately high proteid.

I agree with Dr. Northrup in his remarks about laboratory feeding; I believe one could get better results from laboratory milk than from home milk, even with the addition of cream or top milk; there is too much guess work in modifying milk at home.

DR. FREEMAN.—With regard to high fat feeding, I think much error is caused by ignorance as to what cream is. Many of the books prescribe cream and do not state whether it is cream obtained by centrifuging or not; in other words, whether it is a 40 per cent. instead of a 16 per cent. fat. The use of low fats has been well worked out in Straus's milk in New York City. Straus uses no cream in his modifications at all; the children do well upon it.

DR. CRANDALL.—What was said by Dr. Holt in reference to constipation is important and is what the general practitioner should bear in mind. Not many years ago many physicians seemed to think that babies could not get too much fat. It is interesting to note that every instance he recorded was fed upon one kind of milk, which was a clean milk and its production and distribution was well managed. But I think that 5.5 per cent. fat too high to be used for home modification.

DR. MORSE.—Eczema is often due to too much fat. Another sign which I have not seen mentioned as the result of too high fat percentage, is ammoniacal urine. That the ammoniacal condition of the urine is due to high fats was proved by the fact that the condition ceased when the fats in the food were reduced. The only explanation of this which I am able to give is, that during saponification a large amount of fat takes up an excess of alkali from the intestine. In order to avoid too great a loss of alkali the organism is obliged to manufacture ammonia to take the place of alkali absorbed by the fat in the intestine. The urine then contains much ammonia. It has been my experience that when too much fat is given it is not due to the use of too rich milk, but to the fact that doctors and nurses do not understand what was

meant by top cream and top milk. They do not realize that it makes any difference whether milk has set six, eight or twelve hours, or whether the top 6, 8 or 10 ounces were taken. I believe that much missionary work is to be done in this direction.

DR. ROTCH.—What I have heard makes me believe that the study of fats should be carried on more extensively than it has been in the past. In the intelligent study of the use of fats I think we should direct our attention more to knowing what was given. Young babies have a fairly strong fat digestion. In some cases high fat percentages produce constipation, and in others diarrhea; this I believe to be due to a gradual weakening of fat digestion. Babies may at first have a perfect fat digestion, which can gradually become weakened and develop a series of nervous symptoms. On the other hand, there are cases in which a low fat percentage will give constipation; here the constipation may be obviated by increasing the fat. It is important to know what one is giving and not simply speak of top milk, etc. I advise that in the future, by knowing what is being given, efforts should be made to gain information on this subject and reports made to the Society.

DR. RACHFORD.—I should like to inquire what causes these nervous symptoms in fat feeding; is it not an autointoxication from the intestinal canal?

DR. LA FÉTRA.—With regard to symptoms of fat indigestion the vomiting of quantities of mucus after several feedings is usually connected, I think, with a temporary spasm of the pylorus. In cases of acid vomit with mucus, as in fat indigestion, there is a large amount of this mucus which was formed after, and vomited after, long periods of time. It seems to me that there is an acid stimulation of the muscular layer at the pyloric end of the stomach which causes this vomiting.

With regard to laboratory feeding of babies it has been my experience that home modification, teaching the mother and nurse how properly to modify the milk, is most satisfactory. Personally, I have had no difficulty in getting the milk modified with sufficient accuracy. If the physician is careful and gives written instructions to the mothers and nurses no trouble will be experienced in the home modification.

DR. PUTNAM.—The required information that is so valuable in estimating the high or low percentage of fat can be readily obtained by means of the Babcock tester.

DR. WINTERS.—The cases of convulsions reported by the reader of the paper are not in any way related to high fat percentages in the food. They are typical examples of convulsions due to too frequent feeding. Children of eight and ten months should not be fed every three hours. Children of eight and ten months should have a cereal added to the milk, as milk of sufficient

strength to maintain nutrition at this period is more digestible when combined with a cereal. The constipation from which these children suffered was the result of insufficient proteid for the formation of muscular tissue and the proper development of the muscular wall of the intestine, with the result that there was lack of contractile and expulsive power. Here, again, a cereal would have been invaluable in promoting the laying down of muscular tissue, and in giving bulk to the intestinal contents, which would have promoted regularity of the bowel.

DR. JACOBI.—I have been writing on this subject for forty or fifty years and yet I cannot settle the question for you. In none of the cases reported has anything been done to ascertain the *cause*. In those cases in which indican to any large amount is found in the urine there is proved to be an intoxication. What is it that permits little babies to tolerate so much fat that older babies cannot tolerate? But that is not the fact at all; little babies cannot tolerate fat to such an extent. When babies cannot stand fat any longer they break down; though a baby will stand much hard treatment. If every child that was maltreated died, very few of us would be alive today. Babies six or eight months of age, when they cannot stand the fat any more, break down under the strain.

With regard to constipation while so much fat is taken, the reason is simply this: Insufficiency of the muscular layers. Those babies are fat, rotund and weigh very much; but this is only fat and not muscle; the muscular insufficiency of the intestines cause the constipation, as it does in every rachitical baby. Part of Straus's milk in New York is being made from a prescription I have been teaching these forty years, and here the fat is only a little over 2 per cent.; on this I believe a child will thrive.

Still, I hope this subject will be further studied, especially by Dr. Rachford; clinical observations like those brought up here are good things to know.

AN INVESTIGATION OF THE INFLUENCE OF LABORATORY FEEDING ON 216 INFANTS WITH DISEASES OF THE GASTROENTERIC TRACT, WITH SPECIAL REFERENCE TO THE WEIGHT INDEX.

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In the summers of 1902 and 1903 I was able to study the effects of percentage feeding from laboratory milk in a considerable number of infants of the dispensary class. This unusual opportunity was made possible by the use of the Milk Fund which has now become an established charity contributed to annually by the people of Boston. By means of this fund we have been able for several years to feed during the summer and fall a large proportion of all the sick infants of Boston with acute diarrhea, who have applied to the out-patient departments of the Children's Hospital and of the Infants' Hospital, as well as many from other dispensaries. The number of cases treated in this way has averaged about three hundred each year. In the following series I have, however, included only those cases under one year of age which I have personally observed with the special object in view of studying the influence of a pure milk, accurately modified and appropriately adapted to the individual needs of the patients, together with a series of 65 cases at the Infants' Hospital, whose treatment was for the most part directed by Dr. Charles Hunter Dunn. I wish to acknowledge my indebtedness to Dr. Dunn for permitting me to make use of his records.

Diagnosis of Cases Treated.—The number of cases whose records permitted study was 216. These consisted of 131 cases of fermental diarrhea; 20 cases of chronic gastric and intestinal indigestion; 18 cases of ileocolitis; 15 cases of acute gastric and intestinal indigestion; 9 cases of acute intestinal indigestion; 9

cases of chronic intestinal indigestion; 9 cases of chronic gastric indigestion; 4 cases of acute gastric indigestion and 1 case of severe stomatitis which was primarily responsible for the infant's general disturbance. It is probable that many of the cases classified as fermental diarrhea were in fact cases of ileocolitis. The cases classed as acute, including ileocolitis, fermental diarrhea, acute gastric and acute intestinal indigestion, on the average, were ill seventeen days before applying to the hospital for treatment. The cases classed as chronic, including chronic gastric indigestion and chronic intestinal indigestion had been ill on the average 3.2 months before applying for treatment. It is obvious, therefore, that in many instances the distinction between acute fermental diarrhea and ileocolitis and between chronic fermental diarrhea and the chronic intestinal indigestion was very difficult to make. Again, many of the cases classed as acute gastroenteric disturbances were acute infections superimposed upon chronic gastric and intestinal indigestion.

I have not attempted to express by the terms "infantile atrophy," "marasmus" or "malnutrition," the condition of the infants' nutrition when first seen. With very few exceptions all the babies were decidedly under weight. Fully 25 per cent. of the cases had only attained a weight development of between 40-60 per cent. of the average infants of the same age. Almost all of the cases were first observed in the summer or early fall and were brought in because of the persistence of an acute attack of vomiting or diarrhea. The statistics, therefore, represent not only the influence of good feeding on the weight development of poorly nourished infants, but they show what may and may not be accomplished by proper regulation of the food in poorly nourished infants who are stricken with the acute gastric and intestinal disturbances such as occur in hot weather. That the cases were for the most part seriously ill is indicated both by the weight development of the infants and by the length of time they were ill before applying for treatment. With very few exceptions the hygienic surroundings of the infants remained by necessity the same as before. The only essential change common to all was that of the milk which was supplied to them from the laboratory modified in its percentages and amounts according to the individual requirements.

Weight Index.—To bring order out of a chaos of statistics, I have sought another basis of comparison than that of the number

of grams per week each child gained or lost while under treatment. Obviously such a basis is not wholly fair, for the rate of growth in the average healthy infant varies during the months of the first year. This variation may be expressed as follows:

	Total gain in pounds	Weekly gain in ounces
First three months - - -	5	6
Second " " - - -	3½	4
Third " " - - -	2	2½
Fourth " " " - -	3	3

If, therefore, we are comparing the influence of food on two sick infants, one two months old and one seven months old, we cannot fairly take the average gain per week of the first infant as a standard by which to judge the second, because there is in the infant of two months an inherent tendency to gain weight faster than in the infant of seven months.

The weight of the average infant during the first twelve months of life has been carefully worked out by Dr. Holt. These averages are based on the result of over ten thousand observations on infants who were healthy and digesting normally. The weight curve representing these observations may, therefore, very properly be taken as a constant by means of which we may estimate the percentage development of any infant in comparison with the average healthy infant. To express this relationship between a given case and the average infant I have used the term *weight index,* that is:

$$\text{Weight Index} = \frac{\text{The weight of an infant.}}{\text{Weight of the average infant of the same age.}}$$

If the weight index is multiplied by 100 the result expresses in per cent. the weight development of the infant.

An infant whose birth weight and subsequent growth corresponded exactly to that of the average infant would, therefore, at all times have a weight development of 100, irrespective of the variations in the rate of growth during the four quarters of the first year which have been mentioned above. As long as this percentage development is maintained we know the infant is gaining

proportionately as fast as the average infant. If the gain in weight of such an infant is slower than that of the average child, its weight development becomes proportionately less than 100; if the gain in weight is more rapid than that of the average child its weight development would exceed 100.

If an infant weighs more than the average at birth its weight index is naturally more than 1.0, and its weight development exceeds 100. For example, following the formula given above, an infant whose weight is 8½ pounds at birth has a weight development of 120 per cent. If such an infant should maintain over the average child its advantage in weight development, which it has at birth, its quarterly gains would have to be, as compared with the average infant, 6¼ pounds instead of 5 pounds; 4¼ pounds instead of 3½ pounds; 2½ pounds instead of 2 pounds; and 3½ pounds instead of 3 pounds.

In a similar manner an infant weighing 5 pounds at birth will have a weight development of only 70 per cent. If such an infant should develop proportionately to the average infant, its quarterly gains would be considerably less than the average, that is, it would need to gain only 3¾ pounds instead of 5 pounds; 2½ pounds instead of 3½ pounds; 1¾ pounds instead of 2 pounds; 2½ instead of 3 pounds.

It is evident, therefore, that the number of ounces gained per week are not a fair standard by which alone to judge the progress which is being made in development. If the 8½ pound baby gains the number of ounces per week of the average infant, its percentage development would steadily diminish; whereas, the same weekly gain in the case of the 5 pound baby would cause its percentage development continually to increase.

The weight index enables us to express in definite percentages the relative progress made by infants of different degrees of development. By means of it we may also compare a gain or loss in percentage development of one quarter of the year with that of another quarter, in the same infant, or in two infants of different ages whose nutritive development we are comparing.

In the majority of cases of infants of the dispensary class we can place no reliance on the statements of the parents as to the birth weight. It is, therefore, rare to be able to estimate with any degree of certainty the weight indices at birth. We can, however, weigh such infants when they first come under our observation and so determine the weight indices at the beginning of the treat-

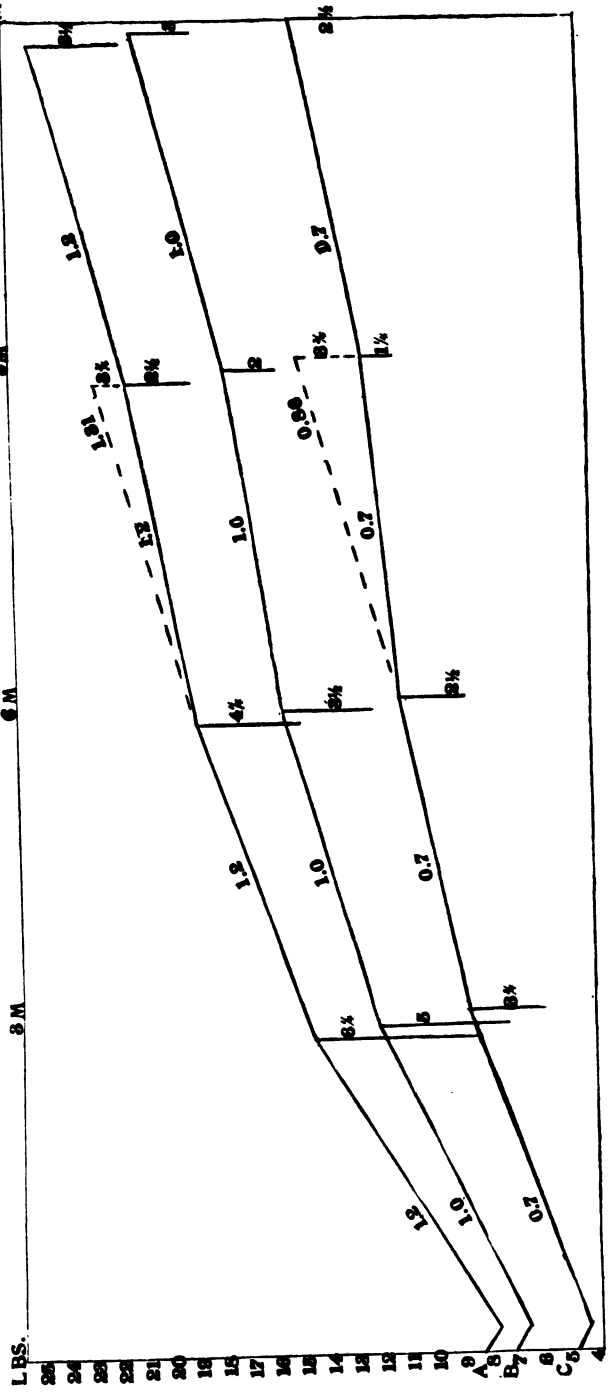

CHART I.—WEIGHT CURVES FOR FIRST YEAR. LINE B REPRESENTS WEIGHT CURVE OF THE AVERAGE INFANT. LINE A REPRESENTS THE CURVE OF AN INFANT WITH A WEIGHT-INDEX OF 1.2. LINE C REPRESENTS THE CURVE OF AN INFANT WITH A WEIGHT-INDEX OF 0.7.

ment. Subsequent weighings will give us the means of estimating subsequent weight indices and of determining and expressing in definite percentages the progress which the infant is making in its weight development.

Chart I. has been plotted to express diagramatically what has been mentioned above.

The middle line "B "represents the weight curve of the average healthy infant, as plotted by Dr. Holt. This curve is taken as the constant by which to estimate the weight index of any infant. The perpendicular lines represent the number of pounds the child gains in the four quarters of the first year. The weight index of such an infant is 1.0, the weight development 100 per cent.

The upper curve "A" represents the theoretical curve of an infant weighing 8½ pounds at birth, with a resulting weight index of 1.2. If such an infant were to maintain throughout the first year the advantage in weight development with which it starts out in life, its quarterly gains in pounds would be as are indicated in the perpendicular lines. These will be seen to vary considerably from those of the average healthy infant represented in curve "B."

In a similar manner curve "C" represents the theoretical weight curve of a 5 pound baby, with a weight index of 0.7, and the perpendicular lines show the quarterly gains in pounds, which would be necessary to maintain throughout the first year the weight development with which it starts in life. These are seen to be considerably less than that of the average healthy infant.

Now, for example, if infant C and infant A each gain from the sixth to the ninth months 3¾ pounds, instead of the theoretical gains expressed in the perpendicular lines, one will find by working out the above formula, that the weight index of infant C has been raised to 0.86, a gain of 16 per cent. in weight development; that the weight index of infant A has been raised to 1.31, a gain of 11 per cent. in weight development. In other words, although each infant has gained the same number of pounds in three months, the smaller infant C has gained proportionately 5 per cent. more in weight development than the larger infant A.

The weight index, then, while not embodying any fixed law of growth, may be used to express in definite percentages the variations in the weight development of an infant, which may occur as a result of natural growth or of disease.

WEIGHT DEVELOPMENT OF THE INFANTS IN THIS SERIES.

The weight indices when the infants were first weighed gives us the best way of showing the extremely low condition of nutrition of the infants in this series when they were first brought to the clinics. These are shown in Table I. Over one quarter of the cases showed a percentage development below 60; one-half of the cases were below 70, and only 3 per cent. of the cases had attained a percentage development of 100 or more. It seems to me that this is a striking commentary on the effects of the ordinary methods of feeding which prevail among the unenlightened poor of our large cities.

TABLE I.

Showing the weight development as estimated by the weight indices of 213 infants, and the groups into which they were divided as a result of treatment.

Weight index in per cent.	No. of Cases.	Percentage.	Group I.	Group II.	Group III.	Group IV.
31-40	1	0.4	1	0	0	0
41-50	12	5.6	8	1	2	1
51-60	43	20.1	24	8	6	5
61-70	54	25.3	28	13	10	3
71-80	39	18.3	22	8	7	2
81-90	38	17.8	16	17	4	1
91-100	19	9.3	8	5	6	0
101-110	3	1.4	1	2	0	0
111-120	3	1.4	1	1	1	0
121-130	0	0.0	0	0	0	0
131-140	1	0.4	0	0	1	0
	213	100.0	109	55	37	12

CLASSIFICATION ACCORDING TO GROUP.

In studying this series of cases, I have divided them into four groups.

GROUP I.—Cases which maintained or increased their weight indices while on the laboratory milk and entirely recovered from the acute gastric and intestinal symptoms for which they were brought to the clinic.

GROUP II.—Cases which showed a loss in weight indices and

yet recovered from the acute gastric or intestinal symptoms and showed material gains in weight.

GROUP III.—Cases which for the most part recovered from the acute gastric or intestinal symptoms while under treatment, but whose weight was not materially increased.

GROUP IV.—Cases which died while under treatment.

The relation of the diagnoses to the above division into groups is shown in Table II.

TABLE II.

	No. of Cases.	Group I.	Group II.	Group III.	Group IV.
Fermental Diarrhea - -	131	68	37	19	7
Chronic Gastric and Intestinal Indigestion -	20	10	5	4	1
Ileocolitis - - - - - -	18	7	3	4	4
Acute Gastric and Intestinal Indigestion -	15	8	3	4	0
Acute Intestinal Indigestion	9	4	3	2	0
Chronic Intestinal Indigestion	9	5	2	2	0
Chronic Gastric Indigestion	9	4	3	2	0
Acute Gastric Indigestion -	4	2	2	0	0
Stomatitis - - - - - -	1	1	0	0	0
	216	109	58	37	12

In the tabulated statistics of these cases, I have arranged, so far as possible, the data concerning the case number in the hospital records, the age, the diagnosis of the gastric or intestinal disease, the number of weeks the infant was ill before being brought for treatment, the weight indices, expressed in per cent., at the first and at the last weighings, the gain or loss in weight indices, the initial loss in weight before the infant began to gain, the total number of grams gained or lost between the first and last weighings, the total number of grams gained from the lowest weight reached while under treatment, and the condition of the patient at the end of the treatment. These tables have been omitted owing to their bulk.

GROUP I.

This comprised 109 cases or 50.4 per cent. of all the cases treated. The average length of time each case in this group was

kept upon the milk, that is, the time between the first and last weighings, was 8.5 weeks. The shortest length of time for a single case was one week, and the longest was thirty-one weeks. The average gain in weight index, expressed in per cent., was 8—none of the cases fell below the weight index when first observed. In 1 case the weight index was raised from .59 to 1.15, or a gain of 56 per cent.; in another from .52 to .95, or a gain of 43 per cent.; in another from .51 to .80, or a gain of 29 per cent.; in another from .51 to .79, or a gain of 28 per cent. There were 5 others who gained over 20 per cent.; 14 who gained from 15 to 20 per cent.; 10 who gained from 10 to 15 per cent.; 28 who gained from 5 to 10 per cent.; 35 who gained from 1 to 5 per cent., and 13 who maintained their initial weight indices.

The average gain per week between the first and last weighings was 149 grams. The average gain per week from the lowest point reached to the last weighing was 156 grams.

GROUP II.

This includes 58 cases, or 26.8 per cent. of all the cases treated. The average length of time each case in the group was kept upon the milk was 5.9 weeks. The shortest length of time for a single case was five days, the longest was twenty-eight weeks, but this group was as a whole under treatment for two and one-half weeks less than Group I. The results were very satisfactory in that the recovery from the acute gastric and intestinal disturbance occurred and was associated with a gain in weight in each case. The average weekly gain in weight was, however, only 79 grams, or 94 grams, if estimated from the lowest point reached while under treatment. The gain was not sufficient to maintain the weight indices, the average loss of which was 3.4 per cent., as compared with the gain of 8.2 per cent. in Group I. The cause of this difference, as may be seen by referring to Tables I. and II., was not due to the difference in the kind of disease from which the infants were suffering, nor to the physical development of the infants when first seen. The virulence of the infection or the poorer hygienic surroundings may possibly be a partial explanation, but in my opinion the chief explanation is to be found in the shorter time each infant of the group received the milk.

Groups I. and II., therefore, represent the cases which, all things considered, did exceedingly well. Together they represent

167 cases, or 77 per cent., of the whole. Table I. shows that of the 56 cases whose percentage development, as judged by the weight index, was between 40 and 60 per cent., 33 are classed in Group I., and 9 in Group II. Many of these were to all appearances cases of what would usually be classed as infantile atrophy.

GROUP III.

Group III. includes 37 cases, or 17.2 per cent. The average loss in weight index was 7.2 per cent., or a fraction more than twice as much as the loss in Group II. The average length of time the cases were kept upon the milk was 3.7 weeks, or about two-thirds that of Group II. The average loss in weight per week was 47 grams estimating from the first to the last weighings. If we take into consideration the gain in weight from the low point reached, the average loss was only 6.5 grams per week, showing that the group as a whole had at the end of the 3.7 weeks of treatment almost recovered the initial loss in weight due to the acute gastric or intestinal condition and to the restriction of food necessary to meet the symptoms.

Fifteen of the 37 cases had recovered completely from the acute gastric or intestinal symptoms when taken off the laboratory milk, 11 others were improved, and 11 were not improved.

Letters were written to all cases not well and thriving at the time the laboratory milk was stopped to trace, so far as possible, their subsequent history. Cases that died of the gastric or intestinal disease for which they were being treated soon after the cessation of the hospital treatment have been included among the fatal cases in Group IV. None of the unfavorable cases in Group III. died, so far as could be learned, but as several cases were not heard from in reply, it is reasonable to suppose that some of those classed as "not improved" died subsequently.

GROUP IV.

Of the 216 cases treated 12 or 5.6 per cent. died in spite of everything that could be done in the way of careful feeding and medical treatment. This number includes those cases which began as out-patient cases and, subsequently growing worse, were referred to the house and there died.

Statistics of mortality are sometimes misleading and it will, I think, be worth while to go more into the detail of the classification of the fatal cases.

The purpose of this experimental investigation has been to study the influence of feeding, together with appropriate medical care, upon the nutrition of infants who were suffering from acute and chronic diseases of the stomach and intestines, especially those cases which occurred in atrophic infants. In the selections of cases which could appropriately be fed from the Milk Fund, none was considered too ill to receive the milk; whereas, many infants of good nutrition and with mild attacks of diarrhea were allowed to continue on their previous milk-supply. This series of cases, therefore, represents the most severe cases which the clinics afforded. They were all under one year of age. Certain fatal cases which were fed upon the laboratory milk were not included in the above series, because they afforded no basis of comparison. The fatal cases which were excluded for this reason were as follows: one case associated with severe congenital syphilis and acute otitis media; one with tubercular bronchopneumonia; another with nontubercular bronchopneumonia; one case of tubercular meningitis, and another of general tuberculosis.

There were, as has been stated, some cases in Group III., 8 in number, who were "not improved" at the time of their discharge, but whose subsequent histories could not be traced. It is fair to suppose that some of them died. If we should assume that all of the 8 doubtful cases died, the total mortality would be raised from 5.6 to 9.2 per cent., but this is, of course, an extreme point of view to take.

TABLE III.

Showing a summary of results obtained in the four groups into which the series is divided.

	No. of Cases.	Average length of time in weeks on the laboratory milk from the first to the last weighings.	Average gain or loss in weight index expressed in percentages.	Average gain or loss per week in grams between the first and last weighings.	Average gain per week in grams from the lowest weight reached until the last weighing.
Group I.	109	8.5	+8.2	+149.	+156.
Group II.	58	5.9	—3.4	+ 79.	+ 94.
Group III.	37	3.7	—7.2	— 47.	— 6.5
Group IV.	12	3.7	——	——	——

A few of the important points in connection with each of the fatal cases may be of interest.

CASE I. (1470). This infant was six months old. She was

breast-fed for four months, after which she was given $\frac{2}{3}$ milk and $\frac{1}{3}$ water. One child in the family had died of tubercular meningitis, and another was at the time ill with pulmonary tuberculosis. The baby's weight was 10 pounds, showing a weight development of 65 per cent. There were no positive signs of tuberculosis, but for six weeks the infant had lost in weight and strength. The symptoms beginning as an attack of gastro-enteric indigestion soon developed into those of ileocolitis. After one month's treatment in the out-patient department the case was referred to the house, where it died at the end of another month. No autopsy was allowed. The suggestion of a tubercular ileocolitis was strong.

CASE II. (1480). This infant, premature at eight months, at the end of four months weighed only 7 pounds 2 ounces. She had had an attack of fermental diarrhea two weeks before coming to the clinic. The stools were then normal, but the nutrition very poor, the weight index being only 52 per cent. The child gained one pound in two weeks, and was then suddenly taken with convulsions, followed by diarrhea. In the following two weeks, the gain was only 4 ounces, but the diarrhea improved, then, without any exacerbation of the intestinal conditions, the baby began to lose weight rapidly and died at the end of the sixth week of treatment.

CASE III. (1721). This infant was nine months old when first seen. It had been fed on Eskay's Food for six months, and then on milk and water. The weight on entrance was 14 pounds 6 ounces, or 82 per cent. in weight development. For two months the infant had had a severe fermental diarrhea, which for a week had been associated with bloody stools and had evidently turned into an ileocolitis. The child had the laboratory milk for two days without improvement. It was then sent into the house and died at the end of six days.

CASE IV. (36310). This infant was three months old. It had been fed on condensed milk, malted milk, diluted cow's milk and proprietary foods. It had had intestinal indigestion for eight weeks, with blood and mucus for three weeks. It was said to have been a large baby at birth, but when first seen its weight development had diminished to 57 per cent. In the course of two and one-half weeks the movements had become yellow and fairly digested and six a day. The vomiting had ceased. The

child was, however, too ill to be weighed. It died at the end of the third week of treatment. Bacillus pyocyaneus was found in pure culture in the stools by Dr. Dunn.

CASE V. (35718). This infant was two weeks old. Since birth it had been fed on equal parts of milk and water. The weight when first seen was 2,910 grams (6.6 pounds). It was suffering from fermental diarrhea. It was sent to the floating hospital, where it remained three weeks, losing 380 grams. The baby was kept on the milk for three weeks, during which time the movements had been reduced to two to three a day. There was no vomiting but the baby grew weaker, lost in weight, took practically no nourishment and died on September 3d.

CASE VI. (35725). The infant was ten weeks old and had been fed on condensed milk entirely. He weighed 3,690 grams (7¾ pounds). His weight development was 65 per cent. In two weeks' time he had gained 270 grams. The vomiting had stopped, the movements had been reduced to four a day, and the baby was taking his food eagerly. He was suddenly taken with persistent vomiting, the stools remained the same, his strength failed rapidly and he died on the third day.

CASE VII. (4226). This infant was seven months old and one of twins. For five months it had vomited four or five times a day. The child was sent into the hospital with an attack of fermental diarrhea and on discharge the bowels were in normal condition. Soon after there was a fresh attack of diarrhea, which was allowed to run for a month without treatment, while the weight diminished from 10 pounds 2 ounces to 8 pounds 11 ounces, and the general condition was very poor. There was strabismus, convulsions, rigidity of the neck and apathy. She was again referred to the house, where the meningeal symptoms gradually passed away. The infant was discharged much improved in two weeks, having gained 1½ pounds. The movements were normal. She was then put on a home modification, and in ten days returned with an attack of vomiting associated with rapid loss of weight, which dropped to 8 pounds 5 ounces. Laboratory feeding was resumed, but all treatment availed nothing and the infant was again referred to the house, where it died after an illness of seventeen days. There was no autopsy. The weight development was 61 per cent. when first seen, and 48 per cent. when referred to the hospital on the last occasion.

CASE VIII. (4674). This infant was one of twins. It was

five months old and weighed 3,472 grams (7 pounds). The weight development was, therefore, only 47 per cent. The baby had had whooping-cough when four weeks old. There were suspicious signs of tuberculosis at the right apex. For three weeks the symptoms of gastric and intestinal indigestion had been growing worse and the baby was under observation for one week only before it died.

CASE IX. (4276). This infant was seven weeks old. It weighed 4,590 grams at birth (9¼ pounds), making its weight development 147 per cent. It was fed on condensed milk for three weeks and Eskay's Food for four weeks. At the end of that time the weight index was 77 per cent. It had gastric indigestion and constipation when put upon the Milk Fund. Eight days later it developed a severe ileocolitis and died in the hospital.

CASE X. (3657). This infant was eight months old. She weighed nine pounds at birth, showing a weight development of 126 per cent., which at eight months of age had diminished to 53 per cent. It had been breast-fed for one month and since then had received condensed milk. For eight weeks the baby had had intermittent attacks of diarrhea, accompanied by a severe prolapse of the rectum and painful fissures. The mother refused to leave the child in the hospital and the baby died of exhaustion in about three weeks.

CASE XI. (4244). This infant was nine months old. At birth it was said to have weighed 5,456 grams (11 pounds), making its weight index 1.54 or 154 per cent. It had been fed on condensed milk for two months, and after that on Eskay's Food, and when first weighed showed a weight index of only .55 or 55 per cent. It was brought to the clinic for fermental diarrhea of one week's duration. The character of the movements improved, the child gained 4 ounces in three days, then began to vomit and refused food. It was referred to the house, where it remained for two weeks without improvement, was discharged against advice and died soon after.

CASE XII. (4466). This infant, seven weeks old, was said to be a large baby when born. Its weight index when first weighed was .74 or 74 per cent. It was breast-fed for two weeks. It had a fermental diarrhea of two weeks' duration, during which it had been given gum arabic and water. There was a chancroid on the vulva and also a gonorrheal vaginitis. The baby was treated for

two weeks, but no improvement occurred. It was learned that the child died soon after treatment was stopped.

GENERAL METHOD OF TREATMENT.

The general method of treatment pursued in the management of these cases was the same in both clinics. Very few of the cases of diarrhea were seen in the first twenty-four or forty-eight hours. If the movements were green and offensive, containing much undigested food, mucus or blood, a laxative either of castor oil, milk of magnesia or calomel, $^1/_{10}$ grain for ten doses, were usually given and in many cases this was supplemented by irrigation of the large intestine, two, three or four times at the beginning of the treatment, but rarely for a longer time than two to four days. Stomach washing was employed in not more than 2 or 3 cases. In almost every case 3 to 5 grains of bismuth were given before each feeding until the movements had been reduced to three or four a day and were of proper consistency and well digested. In some cases with very watery movements an astringent, usually tannigen, in ½ to 2 grain doses were combined with bismuth. Small doses of brandy were prescribed between the feedings in cases which were very weak.

The routine use of barley-water or rice-water until the stools became normal was purposely avoided. It was our opinion that the nutrition of the infants so treated is often unnecessarily taxed by prolonged starvation. A 1 per cent. solution of starch cannot be considered to have much nutritive value. Its chief value lies in the fact that it provides an infant with a sterile fluid and water sufficient to counteract in part the drain upon the tissues as a result of the watery discharges. We believe that a practically sterile fresh milk, if such can be obtained, with greatly reduced percentages of fat and proteids, is quite as efficacious and of far greater nutritive value, after the initial rise in temperature, than the prolonged use of barley-water. As has already been stated the average length of time the acute diarrheas were allowed to run in the series before the infants were brought to the clinics was seventeen days. Sometimes it happened that the infants had during their illness been kept upon cereal waters, and still the diarrhea persisted. In such cases, and in all cases of prolonged diarrhea, the exhaustion produced by the lack of nourishment is often a factor in preventing an infant from combatting the influence of

disease. We may starve the bacteria, but we may also starve their host and it is often a nice point to determine whether the benefit incurred by the one is not offset by the injury to the other. I am not attempting to argue that the use of cereal waters for twenty-four or thirty-six hours in the beginning of treatment is deleterious, but I wish simply to raise the question, If their prolonged use until the stools become normal, as one often sees in practice, does not tend to reduce an infant's strength and nutrition to a point which can be avoided with safety and with decided advantage to its nutrition?

The general principle followed in regard to the feeding of all the cases in this series was to begin as soon as practicable on a very weak milk, usually writing a prescription calling for 2 per cent. of fat, 5 per cent. of sugar, 0.25 per cent. whey proteids and 0.25 per cent. of caseinogen. In very young babies the fat was sometimes reduced to 1 or 1.5 per cent. If the diarrhea had been running for several days, and the baby showed signs of exhaustion, the parents were given emergency prescriptions which they took at once to the laboratory and received a basket of milk, the feedings of which were begun that afternoon. In these cases I usually directed that the food should not be given until after the laxative had acted, and after one irrigation had been administered. If the case was not very urgent, or if the diarrhea was of recent occurrence, the mothers were directed to stop all other kinds of food and, until the laboratory milk was received next day, to give only brandy and water, or plain boiled water or barley-water. In a few cases associated with severe vomiting, the use of barley-water or albumin-water or whey for thirty-six to forty-eight hours, preceded the milk prescription. During the hot weather the milk was invariably heated to 155°F. The percentage of lime-water varied from 5 to 15 or 20, according to the indications.

After the first prescription calling for milk of the above-mentioned composition, the subsequent treatment simply followed the general principles of the application of percentage combinations to individual requirements. It was my experience that the element of milk, which an infant finds most difficult to digest in these acute and chronic diarrheas, was the caseinogen. We were, however, generally able to raise rapidly the whey proteids to the maximum amount of .90 per cent., keeping the caseinogen at 0.25 per cent. When the stools had been reduced to three or four a day and were of a fair consistency and fairly smooth, the fats and the caseinogen

were gradually raised, even if a small amount of mucus was still present in the movements. The rapidity with which the fats could be raised depended upon the amount of gastric indigestion associated with the diarrhea. In the diarrheas without vomiting the fats were raised much more rapidly than in these cases in which there was both gastric and intestinal indigestion. If the fats were raised too rapidly, they caused either regurgitation or vomiting, loose movements with small lumps of undigested fat, easily mistaken for curds, or a cessation in the gain in weight. If kept too low the movements lacked sufficient bulk and the infant either lost in weight or the gain was too little.

In the beginning of the treatment of a new case we insisted upon very frequent attendance at the clinics as often as two or three times a week, and at each visit, usually, the infant was weighed. The result of the weighing gave us indications for the change in the food no less important and valuable than the data obtained by the examination of the stools. The importance of very frequent visits will be appreciated by all, for the harm which comes from keeping an infant too long on a food, too weak or too strong, is daily impressed upon us by the cases of mothers, who, in spite of warning and instruction, neglect to keep their infants under close observation.

In a general way our object was to work each infant up to the basis on which the healthy average infant of its age should be fed. In doing this I feel that the use of whey as a means of raising the nitrogenous percentage of the food at a time when the power of the infant to digest caseinogen is slight, is of great assistance. One per cent. of nitrogen in the form of whey probably does not have the same tissue building power as 1 per cent. of nitrogen in the form of caseinogen. In these cases of difficult digestion, however, it is not a question of the abstract food values, but one of digestion and of absorption; and there is no question, I think, that in many sick infants we can by the use of whey double the amount of proteids that could otherwise be given without interfering with the infant's power of digestion.

The theoretical basis toward which we worked in increasing our percentage combinations from the low initial formula of 2 per cent. fat, 5 per cent. sugar, .25 whey proteids and .25 caseinogen, with which nearly all cases were started, independent of their ages, is seen in the following table:

TABLE IV.

THEORETICAL BASIS FOR FEEDING A HEALTHY INFANT (ROTCH).

Age.	Fat.	Sugar.	Proteids.	Proteids if Split.		Amount at Each Feeding in		Intervals between feedings in hours.	Number of feedings in twenty-four hours.
				Whey Proteids.	Caseinogen.	Ounces.	Cubic Centimetres.		
Premature....	1.00	4.00	0.25	0.25	0.25	⅛–¾	4–25	1–1½	24–18
	1.50	4.50	0.25	0.50	0.25		4–25		
At term.....	2.00	5.00	0.50	0.50	0.25	1	30	2	10
End of 2d week.	2.50	5.50	0.50	0.50	0.25	1½	45	2	10
End of 3d "	3.00	6.00	0.75	0.75	0.25	2	60	2	9
End of 4th "	3.50	6.50	1.00	0.75	0.50	2½	75	2	8
End of 6th "	4.00	7.00	1.00	0.90	0.60	3	90	2½	8
End of 8th "	4.00	7.00	1.25	0.90	0.75	3½	105	2½	7
End of 12th "	4.00	7.00	1.50	0.90	1.00	4	120	2½	7
End of 4 months.	4.00	7.00	1.50	0.75	1.25	4½	135	2½	6
End of 5 "	4.00	7.00	1.75			5½	165	3	6
End of 6 "	4.00	7.00	2.00			6	180	3	6
End of 8 "	4.00	7.00	2.50			7	210	3	6
End of 9 "	4.00	7.00	3.00			8	240	3	6
End of 10 "	4.00	6.00	3.00			8	240	3	6
End of 11 "	4.00	5.00	3.00			10	300	3	5
End of 12 "	4.00	4.75	3.50			10	300	3	5

The data given above are simply a guide to the rules for feeding of the average healthy baby; they are only approximate and not intended to be followed in cases of difficult feeding without due consideration to the individual requirements.

The influence on the bodily weight of feeding with a very weak modification of a pure milk in the manner described is strikingly shown in Table V. In this it will be seen that in 91 cases, or 44.5 per cent., the loss in weight was immediately checked and the infant began to gain in weight coincidently with the improvement in the gastric and intestinal condition. In a little over half the cases the loss of weight continued for a short time, as the amount of food ingested was not sufficient in strength and quantity to maintain the weight equilibrium of the body. In 178 cases, however, or 87 per cent. of the whole, the initial loss of weight did not exceed 248 grams, or ½ pound. This initial loss in weight usually occurred in the first two or three days, and in the great majority of cases was quickly regained.

TABLE V.

SHOWING THE INFLUENCE OF FEEDING IN CHECKING LOSS OF WEIGHT.

		No. of Cases.	Per cent.	
No initial loss in weight	91	91	44.5	87 per cent.
Initial loss of 1-124 G (¼ pound)		50	24.5	
" " " 125-248 G (½ ")		37	18.0	
" " " 249-372 G (¾ ")		11	5.5	13 per cent.
" " " 373-496 G (1 ")		8	3.5	
" " " 497-620 G (1-¼ pounds)		2	1.0	
" " " 621-744 G (1-½ ")		2	1.0	
" " " 745-868 G (1-¾ ")		1	0.5	
" " " 869-992 G (2 ")		1	0.5	
" " "993-1364 G (2-¾ ")		2	1.0	
		205	100.00	

Summary and Conclusions.—The influence of laboratory feeding was studied on 216 infants, with diseases of the gastroenteric tract, the great majority of which were cases of fermental diarrhea and ileocolitis. The acute cases were ill on the average seventeen days, and the chronic cases 3.2 months, before they were brought to the clinic for treatment. They were kept upon the laboratory milk for lengths of time varying from one to thirty-one weeks.

As an aid in comparing the influence of the feeding upon infants of different ages and stages of development the nutrition of each infant at the beginning and at the end of treatment was judged by the estimation of its weight development. This was calculated from the *weight index,* which is simply the ratio of the weight of a given infant to the weight of the average normal infant of the same age. Judged by this standard over 50 per cent. of the cases had a weight development of only 40 to 70 per cent. when first seen. As a result of the treatment, the series is divided into four groups.

GROUP I.—Cases which maintained or increased their weight index while on laboratory milk and entirely recovered from the acute gastric and intestinal symptoms for which they were brought to the clinic. This group comprised 109 cases or 50.4 per cent. The group as a whole gained 8 per cent. in weight index, or a

maximum average gain per week of 156 grams from the lowest weight reached during treatment.

GROUP II.—Cases which showed a loss in the weight indices and yet recovered from their acute gastric and intestinal symptoms and showed material gains in weight. This group includes 58 cases, or 26.8 per cent. The maximum average gain per week from the lowest weight reached was 94 grams. This was not sufficient to maintain the weight indices, the average loss of which was 3.4 per cent. This group received treatment on the average for only 5.9 weeks as compared with 8.5 weeks in Group I.

GROUP III.—Cases which for the most part recovered from the acute gastric and intestinal symptoms while under treatment, but whose weight was not materially increased. This group includes 37 cases, or 17.2 per cent. The average length of treatment was 3.7 weeks, the loss in weight index 7.2 per cent., and the average loss in weight from the lowest point reached was 6.5 grams per week. Eleven cases in this group were "not improved," while under treatment. Of these 8 could not be traced and some of them may have died.

GROUP IV.—The fatal cases. There were 12 in number, making a mortality of 5.6 per cent. in the series of 216 cases, the results of which were known at the time the treatment was stopped.

All the cases in the series were sick infants, and the results are not to be compared with those obtained in the feeding of average healthy infants.

The infants were fed upon appropriate modifications of laboratory milk, usually beginning with very weak whey-cream mixtures. This milk was administered within twelve to eighteen hours after the first visit, and was gradually increased in strength. In one-half the cases there was no initial loss in weight following this method; in 87 per cent. of the cases, the initial loss was less than one-half pound (248 grams).

DISCUSSION.

DR. KERLEY.—There must be some difference between the dispensary children of Boston and those of New York. It is impossible for me to feed as high fat to this class of patients as Dr. Ladd has used in Boston. In the dietetic management of the acute intestinal disorders I am not able to resume milk until the temperature becomes practically normal and until the stools approximate the normal as regards character and frequency. When

milk is resumed I never gave it stronger than ½ per cent. fat and ½ per cent. proteid, and 4 per cent. sugar.

With regard to the cases of indigestion I believe that Dr. Ladd would have gotten better results if he had washed the stomach more often. For a few years past I have been accustomed in cases of acute digestive derangements to wash the stomach out daily or every other day; this increases the babies' appetites and their capacity for food. This procedure has been of special value in the cases in which there is no vomiting, the evidence of stomach derangement being loss of appetite, and a very weak food capacity. It is surprising to observe the large amount of mucus removed from a child's stomach in which there has been no vomiting.

DR. FREEMAN.—One of the most important things in Dr. Ladd's paper is the working out of the weight index and I question whether it would not have been better to attempt to show the results by measurements of length rather than by weight. If a child at birth weighs 5 pounds and measures 17 inches in length the weight index should be corrected for that child; or, if the child measures 19 or 20 inches in length the weight index should be more than normal gained by a normal child, because this would be a long but emaciated child.

DR. CRANDALL.—Another factor which should be considered is that well-developed or well-nourished babies at birth frequently do not gain in proportion to those weighing the same and yet not so well developed or nourished. A thin child of large frame often gains very rapidly during the first three months. I should like to ask Dr. Ladd if these large babies, weighing 10 pounds or so, kept up the weight index to the end of the first year. I think that the large babies would incline toward the average and so would not keep up the weight index.

DR. LA FÉTRA.—Dr. Ladd's contribution is of the greatest value and I hope that he will continue his investigations, and go even further and consider both the children of high weight, 8½ or 9 pounds, and those of low weight. It has been my observation that small babies, those born prematurely or even at term and weighing 4, 5 or 6 pounds, will increase in weight more rapidly than those born at term, weighing 7½ or 8 pounds, and at the end of one year they will attain a weight more than three times their birth weight. Whereas, larger children, as a rule, will not increase so rapidly. A baby weighing 10 pounds at birth will seldom weigh 30 pounds at the end of twelve months; whereas, a baby weighing 7 pounds will frequently weigh 21 pounds at the end of that time. I think a norm should be established for both very large and very small babies.

With regard to the treatment of gastrointestinal affections in summer, at the Vanderbilt Clinic in New York, I have the mothers use cereal waters at first; later I often make use of one of the proprietary foods during the early period of treatment and before

milk is resumed. Barley-water and the proprietary foods, are low in fat. In all these cases I believe that when milk is resumed the fat should be kept very low, but as soon as the temperature disappears the fat can be gradually increased, as well as the proteids, until the normal ratio is established between the fat and proteid for a child of given age. Dr. Holt has pointed out that during the first three or four months the healthy infant's capacity for fat is comparatively great, and the child can take three times as much fat as proteid. Later on the capacity for fat becomes less, being but twice as much. At the end of a year the proteids should be from 2.5 to 3 per cent. and the fat not much greater.

With regard to laboratory feeding of babies it has been my experience that home modification, teaching the mother and nurse how properly to modify the milk, is most satisfactory, even with Dispensary patients. Personally I have had no difficulty in modifying the milk with fair accuracy. If physicians are careful in their instructions to the mothers and nurses no trouble will, as a rule, be experienced in obtaining a modification that is satisfactory.

DR. HOLT.—The reason such a difference of opinion has been shown is because the members are talking of two different things. There are certain conditions in which the milk should be stopped at once, and often for a long time. Again there is a class of cases in which the milk should be stopped for one, two or three days, during the time of the acute symptoms and which could soon go back to milk mixtures. In some forms of summer diarrhea children are often kept from the milk too long. In cases where there is present an acute intoxication, often due to conditions called "milk poisoning" the milk must be withheld until the children are almost well. In others there is sometimes simply diarrhea from indigestion due to hot weather, and in others to the dysenteric organisms. It is in the last two classes of cases especially that milk, properly modified, might be allowed after acute symptoms.

DR. LADD.—In answer to Dr. Freeman, I did not take any measurements at all. The subject I referred to was the weight and had nothing to do with the length of the infants. I was much interested in the use of a very weak modification of laboratory milk, especially in the whey cream mixtures, in checking the diarrheas, and in their influence upon the initial loss in weight. I wish to call attention again to the fact that there was only a small loss in weight in a large proportion of the cases so treated, fully 87 per cent. of the cases losing not more than ½ pound. Unquestionably babies get well by other methods. I think that possibly some of the failures that result from giving these weak combinations of milk in the acute and subacute diarrheas might be due to inaccuracies of modification or differences in qualities of the milk. A weak modification of a stale milk will not accomplish the same results as that of a fresh clean milk.

GASTROINTESTINAL TOXEMIA.

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Gastrointestinal toxemia as differentiated from autointoxication is a systemic intoxication, produced by poisons formed in and absorbed from the gastrointestinal canal. Autotoxins, excreted into and afterward absorbed from the intestinal canal, cannot, therefore, be classed as intestinal toxins.

Our knowledge of the toxins, which contribute to intestinal intoxication, is not as yet upon a very satisfactory chemical basis. We know, however, that the intestinal contents, even under normal conditions, are toxic, and we know, also, that under certain pathological conditions, the toxins produced by the bacterial fermentation of foods in the intestinal canal very greatly increase this toxicity, with the result that a profound systemic intoxication is produced. Our present knowledge, however, is not as yet sufficient to make it profitable for us to attempt to differentiate clinically, between the symptom groups produced by normal and abnormal intestinal toxins.

INTESTINAL TOXINS.

The poisonous albumoses, which are the intermediate bodies formed in the digestion of albuminous food stuffs, may be mentioned as contributing to the toxicity of the intestinal contents, but these bodies, under normal conditions, are robbed of their toxicity, in their passage through intestinal epithelium and liver cells, during the process of absorption. The body is, therefore, carefully guarded against intoxication from this source. It may be, however, that when these protective mechanisms fail, through disease or functional disturbances of these filtering organs, these bodies may act as toxins.

Bacterial fermentation is the great source of intestinal toxins. It is possible that the poisons produced by bacteria in the intestinal canal may have their origin from three different sources.

(1) The components of dead bacteria may furnish a certain

amount of proteins, some of these bodies we know to be poisonous, as for example, tuberculin, which is a protein of the tubercule bacillus. The bacillus of glanders, and other bacilli also contain poisonous proteins, and it is not impossible that poisons of this type may at times be a factor in producing intestinal toxemia. The rôle, however, which these bodies play in intestinal toxemia has not as yet been determined, and is probably not very great.

(2) Living bacteria in the intestinal canal, may and do excrete ferments or toxins, capable of producing the most profound nervous symptoms. These specific toxins of bacteria are believed to be very potent factors in producing intestinal toxemia. It is evident that the character and virulence of these toxins will depend largely upon the microorganisms producing the fermentation, since certain microorganisms are capable of eliminating much more virulent toxins than others. Whether all of these cell toxins belong to the ferment class is a question as yet undecided. Pathologists, at the present time, incline to the view that the specific toxins are closely allied to, if not identical with, the ferments.

(3) Substances produced by bacteria from the culture media are possibly the most important source of intestinal intoxication. Among the poisons of this class, the ptomains probably hold the most important place. These basic compounds, resembling the alkaloids in chemical structure, are capable of producing the most severe systemic intoxication. The virulence of the ptomain formed depends not only upon the microorganism, but also upon the character of the food material in which it is growing. Certain food materials, such as cheese, milk, meat, and other albuminous foods, when acted upon by certain bacteria, may become very poisonous owing to the development of poisonous ptomains.

While ptomains are the most poisonous, and so far as the etiology of intestinal toxemia is concerned, the most potent of the transition products produced during the process of putrefaction, yet there are a large number of other bodies produced in this way, which may also be factors in producing intestinal toxemia. Among these bodies may be mentioned, indol, skatol, phenol, cresol, leucin, tyrosin, ammonia, sulphuretted hydrogen, volatile fatty acids, oxalic acid, uric, and the xanthin bodies. Of these bodies, however, it must be said, that indol is probably the only one, whose etiological relationship to nervous symptoms has been satisfactorily demonstrated. Herter and others have recently

shown that indol, one of the most constant and readily absorbed products of the bacterial fermentation of albuminous food stuffs in the intestinal canal is toxic, and capable of producing headache, and increased nervous excitability. It is not improbable, therefore, that this body may at times, under the conditions named, contribute to the production of these symptoms.

Indirect Etiological Factors.—Constipation is the most important predisposing factor of both acute and chronic intestinal toxemia. It acts by retarding the passage of food materials along the intestinal canal; this gives time for hyper-fermentation, and for the production of toxins by bacterial action on the intestinal contents.

Too much food, indigestible food, and food that has already undergone bacterial fermentation, may also be important factors in producing, or in prolonging, either an acute or chronic intestinal toxemia.

Lack of fresh air and exercise are also important predisposing factors.

The digestive idiosyncrasies of the individual are predisposing factors which manifest themselves not infrequently. These idiosyncrasies are sometimes made manifest by the taking of such foods as milk, eggs, oatmeal, shell-fish, strawberries, acids and wines. One of the most common of the nervous syndromes developed in this way is urticaria. This symptom group is not uncommonly associated with an acid fermentation in the intestinal canal and may in certain instances be classed as an acid intoxication of intestinal origin.

In studying the etiology of intestinal toxemia, it is necessary to bear in mind the important rôle which the liver plays in protecting the body against intestinal toxins. These bodies can reach the general circulation, and thereby produce systemic intoxication, only by passing through the liver; and in leaving the intestinal canal these bodies pass through the portal vein, and thence through the liver to be worked over by the liver cells, and filtered through by way of the hepatic vein, and lymphatics to the general circulation. In passing through the liver, however, these intestinal poisons are under normal conditions rendered harmless. This socalled filtering function of the liver stands guard between the poisons absorbed from the intestinal canal, and the general circulation.

One may suppose that as long as the toxins in the intestinal

canal are not greatly increased in quantity, and as long as the filtering function of the liver is intact, the intestinal toxins cannot produce systemic intoxication, and one may also suppose that systemic intoxication may be produced by failure on the part of the liver to perform its function of filtering the poisonous blood in the portal vein, or from a great excess of intestinal toxins, which may so overwhelm the liver that these poisons find other channels of entrance into the general circulation.

It is well to keep these facts in mind, since in some instances the liver itself may be primarily at fault in chronic intestinal toxemia. This organ, therefore, must be kept under supervision in the treatment of all chronic neuroses, which are supposed to be either wholly or partly caused by intoxications from the intestinal canal.

Chronic intestinal toxemia may also be an indirect cause of nervous symptoms, by the profound changes it sometimes produces in the blood, contributing as it does to the production of chlorosis, and other forms of chronic anemia. These anemic conditions may in turn cause malnutrition of nerve centres, and in that way produce chronic nervous irritability, and the long chain of nervous symptoms which are not uncommonly associated with profound anemias.

Chronic appendicitis, especially in the adult, is not an uncommon cause of intestinal toxemia. This condition may exist for a long time without being marked by distinct attacks of acute appendicitis. In these cases the symptoms of intestinal toxemia, such as headache and general nervous irritability, may be associated with a mucous colitis, tenderness in the region of the appendix, an occasional colic, and ofttimes a sensation of weight and discomfort in that region on active exercise.

ACUTE INTESTINAL TOXEMIA.

Acute intestinal toxemia is more common in the infant and young child than it is in the adult. This greater susceptibility to the acute forms of intestinal toxemia is probably due to a number of causes, the most important of which is the great irritability and the immaturity of the nervous system of the child. This instability of the nervous system of the child makes it possible for slight disturbing factors to produce maximum results. A small quantity of poison absorbed from the intestinal canal may, through its action on the susceptible nervous system, produce

high fever, convulsions, and other pronounced nervous symptoms, while the same quantity of poison might be easily resisted by the stable nervous system of the adult.

It is also true that severe albuminous fermentations capable of producing virulent toxins are more common in the child than they are in the adult. This may be due to the fact that the hydrochloric acid function of the stomach is not as well developed in the young child as in the adult, and therefore not capable of exercising the same control in preventing intestinal fermentations. It may also be possible that the filtering function of the liver, which is at all ages one of nature's safeguards against intestinal toxemia, is not as well carried out in the child as it is in the adult. But whatever may be the explanation, acute intestinal toxemia is much more common in the young child than in the adult.

While our knowledge of the poisons which produce acute intestinal toxemia is very unsatisfactory, yet there is no doubt but that such a toxemia exists, and that it is one of the most important factors in producing nervous symptoms in the young child, and a somewhat less important factor in producing nervous symptoms in the older child, and the adult. Acute intestinal toxemia occurs so commonly in the infant and young child that we are justified in suspecting this cause, where no other apparent cause presents, as a factor in producing sudden rises of temperature, and acute convulsive disorders.

The nervous symptoms which result from acute intestinal toxemia, may vary in severity from a slight fever, with exaggerated reflexes, to a high fever and convulsive disorder so severe as to produce death. Every physician recognizes the importance of acute intestinal toxemia as an etiological factor in the production of dangerous nervous symptoms in the infant and child, and everyone has seen these convulsive disorders followed by high fever and unconsciousness, quickly relieved by cathartic medication, and cured by intestinal antiseptics and diet.

CHRONIC INTESTINAL TOXEMIA.

Familiar as we are with acute intestinal intoxications, we are slow to recognize the importance of chronic intestinal intoxications, which are produced no doubt by the same intestinal toxins, absorbed in smaller quantities and over a longer period of time.

As the child gets older and the nervous system develops, and acquires greater powers of resistance against these toxins, then

the acute intoxications become less, and the chronic intoxications more important, so that in the older child, these severe forms of acute intestinal toxemia are infrequent as compared with chronic intestinal intoxications.

It is the chronic form of intestinal intoxication to which I wish to call special attention, because its great importance as an etiological factor in producing nervous symptoms in children is commonly overlooked.

Chronic intestinal toxemia may be associated with diarrhea, but is not uncommonly associated with constipation. It must be remembered that constipation may exist even when the child has a movement from the bowels every day, or even two or three movements. These movements may be fragmentary and therefore incomplete, or they may be hard and dry showing that they have remained in the intestinal canal twenty-four or more hours longer than they should.

In order to prevent hyperfermentation, and increased absorption of intestinal toxins, the food materials must not be retarded in their passage through the intestine, and when ejected should be moist and have the appearance and form of the normal intestinal evacuation.

So important, do I believe, is the rôle played by chronic intestinal toxemia in the production of neurotic diseases in the child, that I invariably begin the treatment of nervous diseases whatever the symptom group may be, by a careful investigation of the intestinal canal, and throughout the treatment of these cases I give careful attention to any abnormalities of digestion.

The child should be fed upon food carefully adjusted to its digestive capacity, and there should be no retardation of food stuffs in their passage through the intestinal canal.

Chronic intestinal toxemia is probably a factor in the production of a large group of nervous symptoms, and may, as I believe, aggravate the symptoms of certain neuroses, which are produced by etiological factors entirely foreign to the intestinal canal. Among the symptoms which may be produced or exaggerated by intestinal toxemia may be mentioned, malnutrition, anemia, headache, general malaise, fever, heightened reflexes, convulsive disorders, restlessness at night, night terror, general nervous irritability, hysterical and neurasthenic symptoms, incontinence of urine, brachicardia, hyperesthesia, paresthesia, ner-

vous anxiety, psychoses, and, in rare cases, a syndrome resembling meningitis.

The following case while somewhat unusual in the character of the nervous symptoms which it presents, may be taken as a good example of neurotic disease produced by intestinal toxemia.

SEVERE NERVOUS SYMPTOMS PRODUCED BY INTESTINAL TOXEMIA.

Boy, age five years, has never been strong, has had stomach and intestinal trouble very frequently during his life. At the present time, he is thin, anemic, and has the appearance of being malnourished. During the past year he has been very nervous, and this nervousness has recently very greatly increased, so that at the present time he is irritable, cries on slight provocation. is very restless at night, and has certain peculiar nervous attacks, which led his mother to seek medical advice. These attacks come on suddenly with dizziness; the boy falls to the ground, and his mother thinks he does not lose consciousness, and is sure he has no convulsive movements. Some minutes elapse before the boy is able to regain his feet. These attacks are followed immediately by severe headache, and more or less nausea. Following these attacks, the boy is put to bed, and soon falls into a profound sleep, which may last some hours. From this sleep he awakes almost or quite as well as before the attack.

He has had in all, seven attacks during the last year, and three of them have occurred within the last two months. These attacks which seem to occupy the border land between migraine and epilepsy have not only increased in frequency, but also in severity.

The family history throws no light on the etiology of these attacks. The father is phlegmatic, the mother is somewhat nervous, but there is no history of neurotic disease in the family.

A careful examination failed to show any reflex factors which might be etiologically related to this neurosis.

The mother says that for a long time the boy has not been normal in his intestinal canal. Diarrhea has alternated with constipation. There has been much flatulency. The boy has a fitful appetite, and craves food he should not eat. He has been fed almost anything, because "he ate so little," the mother thought "it would not hurt him."

This history strongly suggested the probability that the nervous symptoms might be due to intestinal toxemia.

An examination of the urine, which was high colored, and had a specific gravity of 1023, showed no albumin, no sugar, but a marked excess of indican.

Treatment.—A dose of castor-oil, followed by a diet, carefully regulated to suit the patient's digestive capacity; active life in the open air; a diastase and iron preparation after meals. Under this treatment the boy's general health gradually improved. He gained slowly in strength and weight. His general nervous irritability, which was greatly improved from the beginning of the treatment, gradually disappeared. He never had any of his peculiar attacks after the treatment was begun. Three months later he was discharged well.

Intestinal toxemia, acute and chronic, is not an uncommon complication of other diseases, such as typhoid fever, malaria, tuberculosis, and chronic diseases of the gastrointestinal canal.

As a complication this condition is probably met with more commonly in the convalescence from typhoid fever than in any other disease. The physician's unjustifiable fear of cathartics in this disease is ofttimes prolonged into the period of convalescence, and as a result, fecal accumulations occur, which result in an intestinal toxemia which prolongs the period of convalescence many weeks.

The urine furnishes valuable evidence of the existence of intestinal toxemia, and ofttimes our attention is called to this condition by the presence of an excess of indican, and the ethereal sulphates in the urine. The presence of these substances in the urine is sufficient reason to suspect a hyperfermentation of albuminous food stuffs in the intestinal canal, and also usually means the retardation of these food stuffs in their passage through the canal, but the absence of indican does not rule out intestinal toxemia, since we may have severe forms of intestinal toxemia in which indol is not an etiological factor.

The etiological importance of the vegetable organisms in producing intestinal toxemia, must not cause us to altogether overlook the possible rôle which animal parasites may play in these pathological processes. In the present state of our knowledge, however, it is not possible for us to make any positive statements as to the exact rôle, which intestinal worms play in producing gastrointestinal toxemia. Lynch in Grancher and Comby's "Maladies de l'Enfance" says that intestinal worms may be responsible for a large group of toxic symptoms, such as urticaria,

ringing in the ears, syncope, vertigo, palpitation of the heart, insomnia, mental anxiety, hypochondria, general nervous irritability, fever, delirium, and convulsions.

While this group of severe toxic symptoms may be associated with intestinal worms, it does not necessarily follow that they are produced by the absorption of poisons which have been excreted by them. Lynch himself points out the possibility of these symptoms being produced in part at least, by the secondary fermentations, which the presence of these parasites excite in the intestinal canal, and notes, also, the possibility of severe symptoms being produced, reflexly and mechanically by their action.

Leukart observed that the ascaris lumbricoides excreted a poison capable of producing toxic symptoms. Huber, von Linstow, Chanson, and Raillet, state that this same worm contains an irritating substance capable of producing a toxic effect on the human organism, and a number of other French authors state that this worm excretes an irritating and convulsive substance. Cao, on the other hand, after carefully investigating the subject, concludes that the evidence at the present time does not justify us in attributing toxic symptoms to the action of poisons excreted by intestinal worms.

While there is considerable difference of opinion as to the etiological relationship which exists between animal parasites and intestinal toxins, it seems to be rather generally conceded that these parasites may either directly or indirectly produce intestinal intoxication. In this connection the following case is of interest:—

SEVERE GENERAL CONVULSIONS PRODUCED BY NEMATODES IN THE INTESTINAL CANAL.

I was called in consultation to see a girl, twelve years of age, who had been having convulsions for several hours. She was a strong, healthy German, with no tendency to neurotic disease. After a few days of slight indisposition, on the part of the intestinal canal, she suddenly had a severe convulsion. On my arrival I found that the physician in attendance had been working with her for some hours, and that during this time, she had had a number of severe convulsive seizures. In the intervals between the convulsions she was unconscious and had considerable muscular rigidity.

After controlling the convulsions with chloroform, the urine,

drawn with a catheter, was found to be normal. Sulphate of magnesia was given by the mouth, and by rectal injections. Some hours later a large ball of round worms (ascaris lumbricoides) was passed with a large amount of fecal matter, and very soon thereafter the patient was restored to consciousness, and the next day was well.

There can be no doubt but that this was a case of intestinal toxemia. The child's age, previous good health, and stable nervous system speak against the reflex origin of these convulsions; and I may further state that while it is impossible for one to exclude absolutely other causes of intestinal intoxication, the discharges from the intestinal canal, were not of such a character as to indicate that the poisons were produced by the fermentation of the intestinal contents.

DISCUSSION.

DR. JACOBI.—Our predecessors knew of many such cases and the earliest books contained reports of such cases. In those times they were more in the habit of partaking of raw foods. Where raw materials are eaten, as raw vegetables in Germany and Hungary, they will produce a group of symptoms such as described. Irritability of the nervous system was certainly present and I believe that anemia of the brain would produce this, or rather predispose to it. I have frequently thought that such an anemia would cause insufficient secretion of both gastric and pancreatic juices and so interfere with normal digestion. I am glad the doctor spoke of the frequent prominence of indican in the urine in such cases, particularly because it is so very easy to make the diagnosis of it. In a great many cases indican will be found and therefore it is a very important diagnostic factor.

Unless I examine the urine in every case of illness occurring in both adults and children, particularly in every doubtful case, I feel that I have neglected something of great importance. Besides, nothing is easier than to obtain a specimen of urine from a baby. In a number of cases it is sufficient to rely upon cleanliness and the application of a large enough bundle of absorbent cotton to the parts; then, when the child has urinated, with a clean hand squeeze the urine into some clean receptacle. I myself usually rely upon the use of the catheter which is easy of introduction in the smallest children, easier than in most adults. Newly-born boys do not have strictures in more than 1 to 10,000 or 15,000 cases, while in adults it has been suggested that strictures were much more frequent. Some difficulty might be encountered in small girls, but not much if one used the eyes carefully; then

one did not usually miss the urethra orifice. Failure to examine the urine more often is the cause of so many cases being overlooked. That is why recent journals are all at once replete with alleged rare or unique cases, for instance, of pyelitis. I have frequently met with it, and simply because I take the trouble to examine the urine.

Babies are little susceptible to reflex influences until after the sixth or eighth week of their lives. When they have convulsions during the first few weeks of life they mean either accidents to the nervous centers or a scanty development; as soon as five or six weeks have passed the reflex rapidly grows and inhibition is but little marked; this accounts for so many cases of convulsions during the earlier period of life. During the first few years of life these reflexes are greatly developed. I believe this to be the reason why intestinal irritation and toxemia are so dangerous in young children; both may be caused by either constipation or by diarrhea.

DR. FORCHHEIMER.—I have been for many years interested in this subject and have made many investigations in connection with it, especially because there were many men who did not believe in the existence of such a condition as an etiological factor. Dr. William Osler was at one time of this number. I have tried to prove the existence of such a condition. Bouchard, in a beautifully written and very interesting work established certain forms of chronic intoxication, intestinal in origin, and he believed he had shown the urine to be toxic in such cases. In order to prove Bouchard's work and to satisfy myself that his deductions were correct, I made a number of experiments taking the urines from patients of different classes. The story is a long one and has already been published. As a result of my experiments I found that the toxicity of the urine used by Bouchard was due to faulty methods; when proper methods were used none of the urine was found to be toxic. So, to determine this toxicity one must turn to other methods. Soon indican came to the front again and, as a result, this was examined for in all cases in which there was a diagnosis made of chronic intestinal autointoxication. Unfortunately, indican itself does not show very much as far as chronic intestinal intoxication is concerned. In a large number of cases no indican is present in the urine; at all events, finding indican once in the urine means nothing. But if after repeated examinations indican is found, it is presumptive evidence that there is an abnormal condition in the intestines, causing a change in the albumin with an end product of indican. So far as an examination of the blood is concerned in cases of chronic intestinal autointoxication there is nothing positive determined. I have come to the conclusion, though, in the examination of a series of cases that there is a slight increase in the eosinophiles.

I rely more upon the clinical evidences of chronic intestinal

intoxication, especially in adults, than upon anything else. In children there is an accumulation of fecal material in certain parts of the intestine, such as the sigmoid flexure, and it will be retained there for some time; this accounts for the constipation and for the diarrhea; when it has not been removed there is constipation; when it remains for any length of time it will produce a diarrhea. In adults the conditions are different. In adults, the deposit of fecal material is at different places and at various curvatures, viz., sigmoid flexure, the hepatic flexure, the splenic flexure and the head of the colon. Sometimes the whole of the large intestine become blocked up.

I have laid stress in diagnosis upon a condition I once described as chronic ulcerative stomatitis which occurs in children quite rarely and in adults very frequently in the form of Rigg's disease. Curiously enough in Rigg's disease I have always found uric acid, and I have come to the conclusion in common with a number of other observers that this is due to autointoxication. This condition in the mouth is not so common in children, but when it does occur it gives very valuable evidence of the existence of this condition in the intestines.

DR. NORTHRUP.—I wonder whether Dr. Rachford can throw any light on the intestinal conditions that occur in children who suffer from pneumonia? Are they due to the eliminations of toxins from the intestine?

DR. RACHFORD.—The significance of indican in the urine, admits of but little difference of opinion. When indican is found in excess in the urine it always means a hyperfermentation in the intestinal canal with the formation of indol and other toxins. It is, therefore, a certain indication of intestinal toxemia, since the indol had to be absorbed from the intestinal canal and passed through the body media before it could be excreted as indican by the kidneys, and since indol is toxic, this process must mean autointoxication.

As previously stated the absence of an excess of indican in the urine does not, however, mean that there is no intestinal intoxication without the formation of an excess of indol, and, therefore, without the appearance of an excess of indican in the urine. The relationship, therefore, of an excess of indican in the urine to intestinal intoxication seems plain. When it occurs it is a valuable sign of intestinal intoxication, but its absence does not preclude the possibility of the existence of such an intoxication.

In reply to Dr. Northrup's question as to the explanation of intestinal intoxications as a complication of pneumonia, I think that this complication depends largely upon the age of the child. In very young children, it matters little what the character of the infection is, intestinal complications are common, but they are not specifically related to any particular infection.

NERVOUS EXHAUSTION IN INFANTS.

BY W. P. NORTHRUP, M.D.,
New York.

It was said that a baby of four months, living in a neighboring city, was slowly fading away; that if something was not done for it soon, it would be too late. The history as related by the mother was substantially as follows: For the first three months the baby was a good sleeper and ravenous feeder, particularly happy and strong. It was stated that he was a good-natured baby, would wake from sound sleep and smile, at any hour, day or evening, for callers. It was nursed at its mother's breast and she had milk enough.

My presumption was that the young mother's milk was now failing, that judicious feeding would soon put the child into good condition, the mother would go home happy, and with her my reputation brilliantly enhanced. A naturally strong baby, wet-nursed through the fourth month, seemed to present an easy feeding problem.

The first look at the mother showed a naturally strong, but worn-out, thin and pale young woman. She had recently been through a strenuous horse-show week, trying at the same time to entertain a houseful of friends and to nurse her baby; and she looked it. The baby, too, looked thin, white, but very alert. It jumped at any noise, smiled spasmodically, twitched, had dark circles under its eyes. The mother, with most engaging smile, declared he was the brightest, best-natured and strongest baby in her city. She said he slept all night, and nursed every time she offered him the breast.

After its journey it was, of course, tired but still alert. The mother said: "He was very cunning in the train (seven hours); he never closed an eye, looked at everybody and everything, visited all about the parlor car, entertained everybody and was pronounced the best-natured baby ever seen."

A sunny south room was provided for baby and nurse, the entire third floor constituting the hospital. A baby carriage and Walker-Gordon modified milk were waiting, and I may say the entire domestic and professional plant of my house was at the service of this bit of humanity. Incidentally, I may add, it strongly resembled in appearance one of the average inmates of the Marasmus Room of the New York Foundling Hospital.

At sight, my courage suffered a severe shock. On this case there hung many hopes and incidentally my reputation.

It must be fed, of course. It had for some hours taken but little, some soup from the buffet in the train—just-add-hot-water-and-serve canned soup, you know. A bottle of the most approved modification of the best of New York's milk product was given to the one-third of a year old infant. He engulfed it in the shortest possible time, while all the friends stood in a circle to see the beginning of the miraculous cure—a delivery from fading and wasting. He seemed to want more. His eye was brilliant, his look alert, he was truly something intellectual.

An incidental remark from the mother that he was "keeping this feeding down," enlightened me. He was accustomed, then, to spit up most of his food; she had scarcely finished when a sudden jet of milk, with high muzzle velocity, made the longest record-flight into air. He took 4 ounces and vomited, apparently, 5. He was tired, excited; his stomach had revolted.

The baby was to be weaned from the mother's breast and fed on modified milk. The mother had practically no milk. The first prescription was a low average Walker-Gordon milk modification. After each feeding he habitually spat up little or much. His accustomed nurse took care of him. She was faithful, but not trained; not wise, but willing to obey.

It was gradually discovered that his feet were always cold, that he was most of the time wet, that he took his bottles rapidly, that he kept his food better at night. These were more points of information.

His feet warmed, his feedings given in a darkened room, his nurse soothing him and all his surroundings tranquil, he did somewhat better. He was thought to do better in the air. Central Park was near, and moderately quiet. All efforts to keep him from ejecting some or all of his food failed and really for the moment the situation was discouraging. A week had passed and no real improvement. Indeed, the tiny thing was smaller and had lost his good humor. He wailed night and day, slept hardly at all. The pediatric household was depressed. No results toward cure, reputation going.

At this point a good child's nurse was found, one who not only was trained and experienced, but had some intuitive insight into the needs and comforts of an infant.

The instructions were to keep the baby in Central Park just

as many hours a day as possible, eight hours not being considered too much. Fancy my surprise then on my return home about four o'clock to find the nurse and baby in the sunless backyard, north side of house, and that they had been there two or more hours. I was in an explosive mood, and making straight for the offending parties. Psh-psh-ee-ee-hh, was my greeting; a commanding look in the eye of the triumphant nurse made me pause. The child was getting its first sleep. For three and one-half hours that little moaning, starting, pale and wasted baby slept face downwards, lying across the arms and lap of the nurse. She had found Central Park too noisy. The sounds made the baby jump and wake. Here it was quiet. It would not sleep in its carriage, so she took it on her lap. At last she found it would sleep only in one position, viz.: stomach down. For three and one-half hours the faithful little woman walked or casually sat a moment, gently soothing the worn-out nerves. For three and one-half hours it slept. It was its first good sleep. From that day the improvement began. From that moment the problem was solved. Nerve rest was imperative. The nurse saved the day, instructed the nurse maid, taught the doctor, cured the baby.

The exhibition of a four months' baby with nervous exhaustion, nervous dyspepsia, prostration, cured by a wise nurse, by rest treatment, is worth contemplation by all teachers and practicers of pediatrics.

The recovery, of course, was slow. After three weeks it had gained three-quarters of a pound, was sleeping, taking good, large feedings of whole milk. From my house it sailed for Liverpool, was the only good sailor in the party, is now in rude health, is noisy and marring furniture.

After the diagnosis was made the early history of the infant was reviewed, and is as follows: The baby made a good start in life. After a few months social demands began to wear on the mother, and the milk suffered. In addition to all this was the incessant wear on the child's nerves. Their house was upon a most noisy trolley corner, where the din and clangor was so great that my backyard in New York City was like drowsy Cathay. The infant had many relatives, its full quota of grandparents, a great grandmother, many aunts and uncles. The young pair had hosts of friends, and all of them endowed with an inordinate superhuman fondness for babies. This found expression in waking the infant from sleep, kissing it, jumping it in the air, making

it smile and coo and gurgle. It was an engaging baby and was most of the time engaged. The father had the pleasing habit of entering his own house with a bound, and discharging a war-whoop to announce himself to his young wife. This, of course, put an end to sleep and tranquility for the baby. Then followed a short gallop with the three to four months old infant, after which other affairs led the lord and master thence—unless perchance he met friends. If he did, he brought them in to see and jump and kiss the baby. By this time it was feeding time, if indeed the feeding had not been hastily engulfed in anticipation of the chance invasion.

It was noted as a precocious and pre-eminently cunning thing that this four months' son of the prize tandem driver should sit on his father's lap and lay his little white emaciated fingers on the lines as though driving. His rolling eyes looked out upon the kaleidoscopic mingling of human beings, horses and traps, "in such a sporty way," the mother said. Save the mark! A bulging-eyed marantic infant, starving and wasting, paraded by young, ignorant and proud parents, and calling him "sporty!"

This four-months-old baby was properly born, reasonably well started on the first months of life, nursed by its mother. By the third month it began to get thin, pale, to spit up food and by continuous, injudicious excitement and gradually failing breast food, soon got into a condition of nervous dyspepsia, until it finally was unable to retain and digest sufficient food to allow it to thrive, and was now wasting from starvation.

All this was corrected by quiet, the judicious care of a wise nurse, a new-found, wise, soothing, comforting, quieting little woman, who liked the baby, took it naturally to her arms and enveloped it in such a warm embrace that it sank into a few short startled sleeps; then, finally, stretched out into one long relaxed infant's slumber, from which it emerged with sleepy winks and stretchings, the like of which it had scarce ever known. This first long sleep taught its lesson. Quiet surroundings were indicated. The infant was fed in a dark room. All bells, telephones and unnecessary noises were either muffled or reduced. Cotton was put in the infant's ears. Above all, this wise nurse was given sole charge of the infant. The presence of the father and friends were injurious to the child, the mother was too worn-out and nervous to be anything but harmful, the baby's usual nurse was also exhausted and nervous. The result was that in a fortnight

the baby was feeding on maximum diet, sleeping perfectly and gaining weight.

It has thrived ever since. Last week a letter came announcing the fact that the baby was one year old and had eight teeth, and is "the finest colt in the paddock" (sporty father).

Another case is of much the same history. A young couple from the Southwest on their way to Newport stopped over in New York for a few days and wished to have the baby, five months old, examined in a routine way, to see that it was thriving and that all was being done for it that was possible. This sounds very noble and high-minded, but it really arose from a kind of vanity on the part of the parents. They thought that all, on seeing their infant, must throw up their hands in approval and applause.

Briefly, it was pale, thin, wide-eyed, alert, encircled under the eyes, starting, spitting up. Advice and admonition were accepted, but by no means appreciated. It was as though one were discussing ancient history; nothing said was taken to themselves.

On their return to New York, the child looked a little tanned, a little better, on the whole, but was nervous and spitting up food.

A friend confided to me that every day in Newport this father and mother went along the ocean drive in the crowded hours, in an automobile, with the baby facing forward and witnessing the parade. Not only that, but about half the time the proud father was tossing it, and facing it this way and that way, exclaiming: "See, see the wheels, see, baby, see the horses, *see,* SEE!" It was even noted by the most dull that the mother and baby both looked nervous, starting and wide-eyed.

By the end of the season in Newport, the father was fairly well, the baby nearly worn out, and the mother quite finished. She left in a few weeks for the baths of Bohemia and spent a year recovering from seeing too many wheels and horses. I heard no more of the baby, but doubt not it is alive and a subject for Bohemian baths.

I think we all see certain members of a family, whose nervous systems are peculiarly sensitive to the nagging of other members. I spent a summer where a little girl was quite a painful exhibition of this. The girl was the youngest of three. The older were strong, stamping and tempestuous, fond of noise and confusion and accustomed to late hours. The youngest was thin, pale, quiet, sensitive, had an artistic temperament, was easily annoyed and

teased, a poor sleeper. The older children goaded her without mercy. The parents, wholly oblivious of the situation, scolded her—worse than all, did not sympathize with her. She was like a little despairing, heart-broken thing, her nerves worn out, her sleep and digestion impaired, her only hope that the summer vacation would end and the family get busy enough with their usual home duties to leave her alone.

In February and March we see many cases of nervous exhaustion in school children. They are pale, jerky, choreic, talking in their sleep, sleeping too little, becoming dyspeptic.

This paper is to call attention to nervous exhaustion in infants. In the work of this Society it is desirable to teach the lesson of quiet surroundings, especially of allowing a certain quiet to initiate stomach digestion. It points to the necessity of close, constant watching, and individual-case-studying.

We need not interest ourselves with the mother who takes a year at the baths to recover from the strenuous life of the winter and summer. We should, however, try to protect the coming generation from nervous exhaustion, nervous dyspepsia, sleepless nights and choreic jerkiness before they have cut their first teeth.

DISCUSSION.

DR. COTTON.—I have had the same experience with a girl past nine years old, who came back from her vacation worse than when she left. There is a foolish notion among the Chicago people that they must go away in summer. Time and again the children are brought back more tired than when they went away, especially those with high-strung, nervous temperaments. So much depends upon the environment and habitat, etc., that each case should be decided upon its individual requirements.

DR. CAILLÉ.—Much of the trouble is due to the excessive activity of the times. So far as the management of children is concerned I believe the average physician is much to blame. In my section of the country the hysterical doctor is the most popular man, and, I think, he is much to blame for the conditions Dr. Northrup criticises.

DR. PUTNAM.—Dr. Northrup has spoken of the wonderfully good influences which a nurse is able to exert on a nervous child, but it is worth mentioning that sometimes highly esteemed nurses have a bad influence even on little babies that is equally wonderful, and often not recognized by parents. I am always shy of a nurse who has had the reputation in a former place of staying awake all night with a nervous and restless child, for, as likely as not, it was

the nurse who had kept the baby awake, not the baby the nurse. Under such women babies formerly placid and happy become restless, nervous and sleepless.

DR. ADAMS.—We should beware of the nurse who puts the children to sleep. It has been my experience to come across such an excellent nurse, who came highly recommended, who had influence over babies, and could make them sleep for twelve hours continuously. I protested against this nurse and told the family that the child, only three or four months old, should not be allowed to sleep all night. One night the nurse did not appear and the child was awake nearly all night. This nurse finally admitted administering "knock-out drops." This happened in two instances, one being a white nurse, the other a black one. I would caution particularly against the much over-rated southern "mammy."

DR. FREEMAN.—Besides the general disturbances there are the neurasthenic conditions to be considered, many of which are so difficult of cure. In cases of nocturnal enuresis the mother and nurse have an influence, and the children may often be cured by placing these children in the hands of a trained nurse. Many cases of constipation in infancy are caused by the influence of environment and can be remedied by placing the children in proper environments.

DR. CHURCHILL.—All must be impressed with the growth of nervous conditions in American life in both adults and children. Much can be done to correct this by general direction of the child's life and a careful investigation of all the surroundings and environment of the child. I recall a child, two or three years old, whose parents were neurotic. The child was extremely nervous and slept only from 10 P. M. till 4 A. M. I was the fourth physician to see this child. He looked perfectly healthy, but an examination of the blood and urine showed that the child was anemic and passing a urine deficient in the amount of solids. After regulating the diet and hygiene the child slept better and improved. President Eliot is right when he says that the salvation of the child is in getting him out of the city: he should have as many months away from the city as possible. It is far better to send children away, especially to the seashore, where there is salt air and salt-water bathing—*e. g.*, to the south of Cape Cod. I would advocate the establishment of more common sense schools where there is no competition for prizes. The latter are excellent things to abolish in schools.

DR. NORTHRUP.—It seems as if adults are conspiring to ruin the in-coming school children by offering so many prizes. The children who always sit in the front seats are usually a crop of "choreics," a most distressing sight. The good students usually give a crop of neurotic "jerkers."

A CASE OF INTUSSUSCEPTION WITH CURE BY SLOUGHING.

SUB-ACUTE INTUSSUSCEPTION IN A BABY OF SEVEN MONTHS, WITH ILLNESS OF SIXTEEN DAYS, THE SYMPTOMS SIMULATING ILEOCOLITIS; ON THE SIXTEENTH DAY PROTRUSION OF GANGRENOUS INTESTINE FROM THE ANUS; REMOVAL OF SIX INCHES OF NECROTIC BOWEL PROTRUDING BEYOND THE SPHINCTER ANI; RECOVERY.

BY IRVING M. SNOW, M.D.,
Buffalo, N. Y.

Baby Charles M., seven months; weight, sixteen pounds; nursed well until March 10, 1904, when the child grew pale, had a sub-normal temperature and vomited frequently for three days. On March 13th, the vomiting ceased and the patient began to have a slight fever—100° F. to 102° F. and from fifteen to twenty scanty dysenteric stools a day; the stools were passed with tenesmus, were greenish in color and often contained blood; the urine was scanty and high colored; the patient continually lost weight. The family physician treated the case by intestinal irrigations, and on March 25th was able to introduce a soft catheter some distance into the colon.

I saw the child at 9:15 A.M., March 26th. The baby was thin; temperature 100° F., heart rapid and feeble; the abdomen was distended and tympanitic; no abdominal tumor or tenderness discovered by palpation. During the examination the baby passed gas and a stool containing mucus and fecal matter, followed by the protrusion from the anus of a mass, looking like ragged membrane; its color was pale, like anemic mucous membrane, the edges were fringed. The tumor measured 1½ inches long and ¾ inches wide, and could not be replaced by moderate pressure. A rectal examination showed that the mass traversed the rectum and

emerged from the sigmoid flexure; no bleeding followed the introduction of the finger. The nurse said the bowel had come down in the night, and that she had pushed it back, thinking it to be a prolapsed rectum. The protruding tissue was evidently necrotic intestine, the result of an intussusception.

The baby was taken to the Sisters' Hospital, and placed under the care of Dr. Eugene Smith. The baby arrived in poor condition, with a pulse of 200. The bowel had receded, and could not be discovered by a rectal examination. It was clear that the child's symptoms, vomiting, diarrhea and emaciation for the past sixteen days were due to a subacute intussusception, that a portion of the bowel had become gangrenous and had twice protruded from the anus. It was also certain that at no time had a complete obstruction to the fecal current existed; for gas and feces had been passed every day since the beginning of the illness. The baby was in poor condition for a laparotomy, and there was no urgent indication for operative treatment. The baby was fed and well stimulated.

On March 27th, the patient was much improved and the bowel again protruded from the anus. Dr. Smith pulled down the necrotic gut outside the sphincter, ligated it distal to living tissue to avoid hemorrhage, cut below the ligature and pushed back the stump into the rectum. The portion of intestine removed was about 6 inches long. The ligature in no way interfered with the fecal current, as the intussusceptum had evidently sloughed partly off at the neck, and the intestinal contents passed through an aperture in the invagination alongside the intussusceptum.

The baby was allowed to nurse and steadily improved, although it had a diarrhea for a number of days. The ligated stump and the remainder of the gangrenous cylinder were not discovered; they either remained in the intestine or were passed unnoticed in the stools. The patient is now in perfect health—ninety-six days after the removal of the necrosed-tissue.

The malady simulated an ileocolitis for sixteen days and without a digital rectal examination and on close scrutiny the protruding mass might have been mistaken for a prolapse of rectal mucosa. When on the sixteenth day the nature of the condition became apparent, a laparotomy would have done no good, and the shock of the operation might have killed the child. The removal of the necrotic intestine March 27th had undoubtedly the effect of shortening the baby's illness and probably saving its life.

To await its spontaneous detachment, would be to allow a source of irritation and autointoxication to remain in the intestinal canal to produce pain, abnormal peristalsis, impaired assimilation and increasing prostration. Dr. Herbert Williams, who examined the intestinal slough, was unable to tell whether the specimen was large or small intestine, or whether the exterior was serosa or mucosa, so extensive was the necrosis. This method of cure of an intussusception in a baby a year old or younger, viz., elimination and discharge of the intussusceptum by sloughing or gangrene is excessively rare, and I can find only 4 other recorded cases. To wait for it or expect it involves almost certain death to the patient. Most infantile intussusceptions run an acute course, produce a complete intestinal obstruction and extreme cardiac depression, and terminate fatally unless relieved by operative treatment. The acuity of the symptoms are proportional to the degree of strangulation.

In the rare cases of subacute or chronic intestinal invagination in babies, the lumen of the bowel, although varying in calibre with congestion, is generally sufficiently large for the passage of the intestinal contents, and no symptoms of obstruction are observed. The irritation and congestion cause a plastic inflammation of the serous surfaces in apposition, and adhesions form, rendering a reduction impossible. As to a spontaneous cure by sloughing of the invaginated gut, it is only possible when the continuity of the canal is restored by firm unyielding adhesions before the proximal end of the intussusceptum becomes gangrenous, or if the line of demarcation is below the neck. Gangrene and detachment of the sequestrum are often announced by rapid heart failure, and numerous stools having an excessive fetid gangrenous odor.

Gangrene may commence:—(1) At the point of constriction, the neck of the invagination, in which case, the intestinal sequestrum has a more or less tubular form, either the mucosa or the serosa being turned outside. (2) At the free end of the intussusceptum or at a more or less distant spot from the neck. In this case the necrotic gut is discharged piece-meal, bit by bit, leaving behind an attached remnant floating in the lumen of the gut.

Leichtenstern[1] states that cure by sloughing, if the invagination be in the small intestine, takes place in 10 to 21 days; if in the colon in about 4 weeks, with wide extremes from 3 days to 6 months. In 5 cases under one year old the average time was 9 days. Henoch's case 3 days; Beeston's 4 days; Steinmeyer 7 days;

Cripps' 4 days; Snow 17 days; 3 were acute and 2 subacute. Wiggins states that in one instance, age not given, the bowel became gangrenous in twenty-eight hours after invagination. All authors agree that sloughing is most common in acute cases. After necrosis and expulsion of the gut, the health is not immediately established, there follows long continued disturbance of the intestinal functions damaging assimilation, viz., colic, diarrhea, constipation, dyspepsia, vomiting, marasmus, stricture at the scar, perforation, separation of the upper and lower segments of intestine, imperfectly united at the point of sloughing.

A modern physician dealing with an intussusception looks on operative treatment as inevitable, yet in reviewing the literature, it is surprising to learn how often nature if left to herself effects a cure.

Leichtenstern states that spontaneous elimination by gangrene occurs in 42 per cent. of all cases of intussusception, being greatly influenced by the location of the invagination, which is ileocecal in 20 per cent.; colic in 28 per cent., and in the small intestine in 61 per cent. of the cases.

It is least frequent in children under a year old; they die of pain, inanition, exhaustion, shock, too early for the process of separation to be complete.

Leichtenstern's statistics show that sloughing of the invagination takes place in 2 per cent. of cases in 1st year; 6 per cent. of cases from the 2d to the 5th year; 38 per cent. of cases from the 6th to the 10th year; 40 per cent. of cases from the 11th to the 40th year; 44 per cent. of cases from the 41st to the 60th year; 46 per cent. of cases above 60 years.

Leichtenstern's treatise on intussusception was written in 1873. It is probable that the condition is now recognized at a much earlier stage, and that few physicians allow the invagination time to slough so that the patient dies of a laparotomy or is relieved by it. Of these cases of spontaneous elimination of the gangrenous intussusceptum 40 per cent. ultimately die of sequelæ connected with the intestinal lesions. Leichtenstern states that 15 per cent. of all intussusceptions are permanently cured by sloughing. Treves[2] more conservatively estimates the recoveries at less than 10 per cent.

In J. L. Smith's[3] collection of 52 cases in children 7 recovered by sloughing, 13 per cent.; none younger than 5 years, the ages

being 5, 6, 6, 9, 11, 12, and 12, years, respectively; separation of the invaginated mass occurred in 6 cases, the average time being 9½ days. Holt[4] finds that in 362 cases of intussusception under 10 years portions of the intestine were passed per rectum in 24 cases—6 per cent.; only 2 cases occurred in infants.

I will conclude with a list of the cases of intussusception in babies, a year old or less, terminating in recovery by gangrene and elimination of the intussusceptum.

(1) Snow: history given above.

(2) Henoch[5]: expulsion of 2½ inches of gut on the third day of symptoms of intussusception.

Baby of twelve months sickened February 28th in the evening with vomiting and crying next morning, evacuations of clear blood from the anus, moderate meteorism; symptoms increased until March 2d, when Henoch first saw the case; vomiting had ceased; no fecal passage could be procured; child in collapse, with finger in the rectum the intussusceptum could be felt; after rectal examination there followed a discharge of dirty brown fluid and the expulsion of a necrotic piece of intestine 2½ inches long, its tissue belonging to the colon. During the next three days there were frequent thin fecal stools. On March 6th renewed obstruction, increased meteorism and colicky pain; nevertheless, the appearance was better, pulse stronger, no vomiting, and the nourishment was well taken. Ice was applied to the abdomen and opium given to relieve pain. On March 8th there were eight to ten fluid stools having a gangrenous odor, but no bits of bowel; the meteorism gradually disappeared; there was a copious diarrhea for some days—complete recovery.

(3) Beeston[6].—Expulsion of intestine on fourth day of symptoms of intussusception.

Baby boy seven months old came under observation on the second day of symptoms of intussusception; insufflation of air tried, unsuccessful; on the fourth day the patient had copious stools and passed a piece of fleshy looking tissue, having a fetid odor—no further trouble.

(4) Cripps[7].—Girl seven months old; ill fourteen days with intussusception which hung outside the anus, and was in a gangrenous state; a portion of this protruding mass was removed, and the rest of the slough came away, bit by bit for a month. The patient was discharged from the hospital, cured. Subsequently the child died of scarlet fever, and at autopsy the small in-

testines were attached to the anus. No trace of the ascending, transverse or descending colon: all had sloughed away.

(5) Steinmeyer[8].—Baby six months old—attacked with acute intussusception; on the seventh day of symptoms there appeared in the stools a segment of intestine, 27 cm. long; it was from the ileum, and the ascending colon; recovery.

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DISCUSSION.

DR. CHARLES P. PUTNAM.—I should like to ask if the normal function of the bowel was retained and if fecal matter and gas escaped.

DR. SNOW.—Fecal matter and gas passed every day.

DR. PUTNAM.—The condition producing such symptoms is surely rare. I believe that it almost never happens. I would ask whether it happened in the other cases referred to in the paper.

DR. SNOW.—No such occurrence was reported in the other cases. The case I report was so similar to an ileocolitis, with the passage of mucus and blood, that no one could make a diagnosis. The diagnosis was only made by the protrusion of the invaginated intestine from the anus.

DR. AUGUSTUS CAILLÉ.—What portion of the intestines presented.

DR. SNOW.—It was impossible to tell, but my own opinion is that it was the ileum. The microscopical examination showed muscular tissue; it was also impossible, on account of the advanced necrosis of the tissues, to tell whether it was mucosa or serosa that turned out.

THE RACHITIC HAND.

BY HENRY KOPLIK, M.D.,
New York.

Some years ago my attention was called to a peculiar manifestation of rachitis which existed in some very marked cases of the disease, and which to my mind might easily have been mistaken for syphilitic disease of the bones had not the history of the case been carefully sifted and considered. In these children the symptoms of rachitis as manifested in the bones were very marked, not necessarily in deformity, but in the distorted growth and deposit of bone, especially of the long bones. I have been accustomed to demonstrate the peculiar condition of the hand which I will now describe as due to rachitis, and as a condition not generally noticed either in the text books or monographs on the subject.

Most of the infants and children in whom the rachitic hand could be demonstrated suffered from marked rachitis, accompanied by pain in the bones to such an extent that one would be apt to think of syphilis; but so different was the picture as compared to that of syphilis, that there could be no doubt as to the real nature of the disease in question. One of these cases I have published in my book with an accompanying figure showing the rachitic hand, and in that article I have laid stress upon the appearances as occurring in the shaft, rather than in the epiphyses of the phalanges. Lately in the transactions of the Society of the Diseases of Children, which was part of the Congress of German Scientists, meeting in Cassel in 1903, Siegert published a notice, with portrait, of the socalled rachitic hand.

Since Wegner's work on the Diseases of the Bones of Young Children, it has been established that in rachitis the bones of the carpus and fingers are drawn into the picture. Wegner described this localization of the rachitic process.

I have had repeated occasion to demonstrate this deformity and it seems to me it has escaped the notice of most writers, with the exception of Sobel, who mentions my description of the condition.

The children in whom this is noticed present nothing peculiar,

with the exception of the ordinary rachitic habitus of the most pronounced type. The head, the chest, the extremities all show manifestations of the disease. I show here a portrait of such a hand in a child treated in my hospital service for an intercurrent affection. The history of the case presents nothing peculiar, but for the sake of completeness I include it here.

Male infant, two years of age. *Family History.*—Mother has five other children, all of whom have rachitis. The mother has never aborted. *Previous History.*—There was a difficult labor, head presentation, no instruments were used. The infant was breast-fed for one and a half years, since then has had a generally mixed diet. The child can sit, but cannot stand; is constipated; has had no previous illness, especially none of the exanthematous type.

Present illness began three days ago with a cough, which increased from day to day. No fever, no chill, no cyanosis. Child has lost weight and gradually became worse.

Present Condition.—Fairly well nourished; teeth in diseased condition. Tongue not coated, large submaxillary and sublingual glands. Lymph nodes in the anterior and posterior cervical, posterior occipital, axillary and inguinal regions palpable. Very marked signs of rachitis. The head large and square, epiphyses of all the long bones enlarged. Marked infracture of the clavicle, marked rachitic rosary, with flaring of the lower part of the ribs. The middle of the diaphyses of the phalanges of both hands bowed and thickened, kyphosis of the lower dorsal region of the spine, which is quite flexible. Pigeon breasted chest, lower intercostal spaces drawn in on inspiration. The lungs show a bronchopneumonia in the upper lobe of the right lung, and bronchitis in both. Spleen palpable below the ribs, abdomen prominent, tympanitic, right kidney palpable. The lower extremities, shaft of tibia, and the lower extremities of the bone show signs of marked rachitis. The remainder of the history is not relevant. Child was discharged cured of its pneumonia.

The above history is given simply to show that there was nothing in the case other than the rachitis which could be regarded as a cause of the peculiar condition of the hand. Photographs were taken of the hands and they show very beautifully the bow of the phalanges. An X-ray shows very distinctly the thickening of the phalanges in the same hand. It can be seen by a study of the radiograph that the soft parts are really bowed

RACHITIC HAND, SHOWING BOWING OF THE FINGERS.

by the thickening of the bones of the phalanges and are not the causation of the peculiar conformation of the fingers. In other words, the appearance of the hand is not caused by any fatty tissue which may be present in the fingers. It may be also

X-RAY OF RACHITIC HANDS, SHOWING THICKENING OF THE PHALANGEAL SHAFT.

seen that the fingers of this hand are longer and more tapering than is usual in the hand of an infant of this age. This seems to be due to a laxity of ligamentous structure of the joints of the phalanges. By comparing this radiograph with one given by John Poland in his atlas of a child of the same age, it is apparent that the distance between the extremities of the phalanges which make up the joints is greater in the case of the radiograph of the rachitic than of the normal hand. These facts may account also for the peculiar incurvated appearance of the fingers at the situation of the joints.

692 Madison Avenue.

AN ANALYSIS OF ONE HUNDRED AND EIGHTEEN CASES OF LOBAR PNEUMONIA IN INFANCY.

BY JOHN LOVETT MORSE, A.M., M.D.,

Instructor in Pediatrics, Harvard Medical School; Assistant Physician at the Children's Hospital and at the Infants' Hospital, Boston.

This study was undertaken because of my feeling that many of the commonly accepted views, not only as to the frequency of lobar pneumonia in infancy, but also as to its onset, course and prognosis, were erroneous and not justified by the facts.

During the last nine years about 1,900 cases, both medical and surgical, were admitted to the wards of the Infants' Hospital. One hundred and eighteen of these had lobar pneumonia. Fifty occurred in the first year of life and sixty-eight in the second year. There can be no doubt, I think, as to the accuracy of the diagnosis in these cases, as they were all in charge of and examined by Dr. Rotch, Dr. Wentworth or by me. In a number the correctness of the diagnosis was proved by autopsy. These figures certainly seem to show that lobar pneumonia is not very uncommon in infancy. Only 60 cases of bronchopneumonia, or just half as many as of lobar pneumonia, were admitted to the hospital during the same period. It is probable, however, that this proportion does not represent the real relative frequency of lobar pneumonia and bronchopneumonia in infancy, as only the more severe cases of bronchopneumonia are brought for admission, and those secondary to the two most common causes of bronchopneumonia in infancy, measles and whooping-cough, are, of course, rejected. It does show, however, that lobar pneumonia must be relatively more common in comparison with bronchopneumonia in infancy than is generally supposed.

ONSET AND HISTORY.—The history of the onset and course of these cases before their admission to the hospital was in many cases wanting or very defective. In 77, however, the history was

fairly complete. Vomiting was mentioned as the initial symptom in 7 cases and a convulsion in one. A chill was not mentioned in any case. While vomiting might easily have escaped mention, it is very improbable that a convulsion would not have been noted, and it seems safe to assume that a convulsion was the earliest symptom in but 1 case. These figures show, therefore, that the onset of pneumonia in infancy is less stormy than is usually supposed, and that a convulsion is very seldom the initial symptom. The sudden onset of high fever was rarely noted, but fever was almost invariably mentioned in the history. Drowsiness and apathy were also very frequently mentioned. There was a positive history of cough in 75 of the 77 cases in which there were definite data, its absence being noted in only two. These cases seem to show, therefore, that the onset of lobar pneumonia in infancy is usually not accompanied by very acute symptoms, but that fever and cough are usually first noted, these symptoms often being accompanied by drowsiness or apathy.

PHYSICAL SIGNS.—Movement of the alæ nasi was noted in 56 cases, its absence in 14. No reference is made to it in the others. It is possible that it was present in these cases also, although not mentioned. My impression, however, from seeing the cases from day to day is that motion of the alæ nasi is by no means a constant symptom of lobar pneumonia in infancy.

Cyanosis was noted as being present in 17 and absent in 53 cases. It is very possible, however, that it may have been present at other times in the course of the disease and not noted. Pallor was noted very often. A condition of somnolence or apathy was mentioned very frequently.

The following table shows which lobes were involved and whether they were involved wholly or in part.

One Lobe.	Whole	Part	Total	Per Cent.
Right Upper Lobe - -	16	4	20	17
Right Middle Lobe - -	0	1	1	1
Right Lower Lobe - -	7	12	19	16
Left Upper Lobe - -	14	2	16	14
Left Lower Lobe - -	24	21	45	38
	61=60%	40=40%	101	= 86%

More than One Lobe.

Right Upper and Right Lower Lobes -	7
Right Upper and Right Middle Lobes -	1
Right Upper, Middle and Lower Lobes -	2
Right Lower and Left Lower Lobes - -	5
Left Upper and Left Lower Lobes - -	2
	17=14%

That is, a single lobe was involved in 86 per cent. of the cases and more than one lobe in 14 per cent. The whole lobe was involved in 60 per cent. and a part of the lobe in 40 per cent. of the cases in which the process was limited to a single lobe. The upper lobes were much more often entirely involved than the lower. The relative frequency with which the lobes were involved was as follows:—left lower lobe; right upper lobe; right lower lobe; left upper lobe; right middle lobe. The left lower lobe was involved in more than twice as many cases, however, as any other lobe. In the cases in which more than one lobe was involved, they were involved together or in succession in about equal number.

FEVER.—*Duration.*—The duration of the fever in the 61 cases in which it could be determined varied from 4 to 18 days. The following table gives the number of cases for each number of days:

	First Year.	Second Year.	Total.
4 days	1	3	4
5 "	4	4	8
6 "	3	3	6
7 "	3	11	14
8 "	2	5	7
9 "	4	4	8
10 "	0	4	4
11 "	1	1	2
12 "	0	2	2
14 "	1	2	3
15 "	1	1	2
18 "	0	1	1
	20	41	61

It shows, as would be expected, that 7 days was the most common duration. About half as many lasted 5, 6, 8 or 9 days. A somewhat larger proportion of the cases in the first year were of shorter duration than of those in the second. The average dura-

tion of the fever was 8.1 days. It was 7.7 days in the 20 cases in the first year, and 8.2 days in the 41 cases in the second year, being nearly the same for the two years.

Height.—The highest temperature was 107°F., in a fatal case. The following table shows the highest point of the fever for the two years and for each year separately:

	First Year.	Second Year.	Total.
Highest Temperature.	No. and Per Cent. of Cases.	No. and Per Cent. of Cases.	No. and Per Cent. of Cases.
Over 106°F.	1= 2%	5= 7%	6= 5%
105° - 106°	16=33%	25=38%	41=36%
104° - 105°	14=29%	13=20%	27=24%
103° - 104°	12=25%	16=24%	28=24%
103° or Less	5=11%	7=11%	12=11%
	48	66	114

It is evident from this table that in the vast majority of the cases (84 per cent.) the highest temperature reached was between 103°F. and 106°F. The highest temperatures within these limits were about equally divided among the degrees and were approximately the same for the two years. In 11 per cent. the temperature did not rise above 103°F., and in 5 per cent. it was over 106°F. The proportion of excessive temperatures, that is, over 106°F., was rather larger in the older infants.

Termination.—The temperature fell by crisis in 64 cases and by lysis in 29 cases, giving a percentage of crises of 68.8. The percentage of crises in the first year was 54, and in the second year 77.5, figures consistent with the general opinion that crisis is less common in the first year than in later life. Collapse during the crisis was noted but once in these 64 cases, although it is possible that it may have occurred in other cases and not have been mentioned in the notes. Granting that it escaped attention in a few cases, the frequency of collapse during the crisis is even then much less than would be expected from the general teaching on the subject. A pseudo-crisis was noted in 9 cases. It occurred the day before the crisis in 6 cases, two days before in 1 case and three days before in 2 cases. Pseudo-crises were equally common in both years.

Irregularity.—The temperature was notably irregular in 7 cases. In three others there were remissions of from two to three degrees, and in 11, remissions of from three to five degrees.

These irregularities and remissions of temperature were equally common at all ages.

PULSE.—The following table shows the highest pulse rates and the mortality for each rate, both in the first and second years and in the two years combined. It will be seen from this table that the usual pulse rate was between 150 and 170, 51.2 per cent. of the cases belonging in this class. In 75.8 per cent. the pulse rate was over 150 and in 6.7 per cent. over 180. In 14.3 per cent. it was below 140 and in 23.7 per cent. below 150. No case died in which the pulse was not over 140. The pulse rate, when over 140, seemed to have little or no influence on the mortality. A rapid pulse was no more fatal in the second year than in the first year, except when it was over 180. The case with the most rapid pulse recovered, however.

Pulse Rate.	First Year.		Second Year.		Totals.	
	Number and Per Cent. of Cases.	Number of Deaths and Per Cent. of Mortality.	Number and Per Cent. of Cases.	Number of Deaths and Per Cent. of Mortality.	Number and Per Cent. of Cases.	Number of Deaths and Per Cent. of Mortality.
110–120			1= 1.5 %		1= .8 %	
120–130	1= 2 %		1= 1.5 "		2= 1.6 "	
130–140	6=12 "		8=11.9 "		14=11.9 "	
140–150	8=16 "	5=63 %	3= 4.4 "		11= 9.4 "	5=45.4 %
150–160	13=26 "	5=38 "	24=35.8 "	4=16.6 %	37=31.6 "	9=24.3 "
160–170	10=20 "	4=40 "	13=19.4 "	1= 7.6 "	23=19 6 "	5=21.7 "
170–180	9=18 "	4=44 "	12=17.9 "	5=41.6 "	21=17.9 "	9=42.8 "
180–190	3= 6 "		4= 5.9 "	2=50 "	7= 5.9 "	2=28 5 "
190–200			1= 1 5 "		1= .8 "	
	50	18	67	12	117	30

RESPIRATION.—The following table gives the respiratory rate and the percentage of mortality for each rate during the first and second years and for the two years combined. It shows that in 75 per cent. of the cases the respiratory rate was between 55 and 80, while in 11 per cent. it was below 55, and in 14 per cent. above 80. No case died in which the rate of the respiration was below 55. The rate of the respiration above 55 had no apparent effect on the mortality. It is to be noted, moreover, that 3 of the 5 cases in which the respiration was over 90, recovered. There was no evident relation between the rate of the respiration and the age, nor did the relation of the respiratory rate to the age seem to have any effect on the mortality.

Rate of Respiration.	First Year.		Second Year.		Totals.	
	Number and Per Cent. of Cases.	Number of Deaths and Per Cent. of Mortality.	Number and Per Cent. of Cases.	Number of Deaths and Per Cent. of Mortality.	Number and Per Cent. of Cases.	Number of Deaths and Per Cent. of Mortality.
35–40	1— 2 %		2— 3 %		3— 2.5 %	
40–45	1— 2 "		1— 1.5 "		2— 1.7 "	
45–50	1— 2 "		2— 3 "		3— 2.5 "	
50–55	3— 6 "		2— 3 "		5— 4.2 "	
55–60	10—20 "	5—50 %	14—20.9 "	2—14.2 %	24—21.3 "	7—29 %
60–65	4— 8 "	2—50 "	2— 3 "		6— 5.1 "	2—33 "
65–70	12—24 "	5—41.6 "	19—28.3 "	3—15.7 "	31—26.5 "	8—26 "
70–75	6—12 "	1—16.6 "	2— 3 "	2—100 "	8— 6.9 "	3—37 "
75–80	6—12 "	1—16.6 "	13—19.3 "	1— 7.9 "	19—16.2 "	2—10 "
80–85	4— 8 "	3—75 "	1— 1.5 "	1—100 "	5— 4.2 "	4—80 "
85–90			6— 9 "	2—33.3 "	6— 5.1 "	2—33 "
90–95	2— 4 "	1—50 "			2— 1.7 "	1—50 "
95–100			3— 4.5 "	1—33.3 "	3— 2.5 "	1—33 "
	50	18	67	12	117	30

MORTALITY.—Thirty-one cases died, giving a mortality of 26.3 per cent. Eighteen of these cases were in the first year, giving a mortality of 36 per cent., and 13 in the second year, giving a mortality of 19 per cent.; that is, the disease was nearly twice as fatal in the first as in the second year. Excluding the 4 cases in which death occurred late as the result of empyema, the mortality was 22.8 per cent. for the two years and 31.9 per cent. and 17.9 per cent. for the first and second years respectively. These figures give a very different picture of the severity of pneumonia in early life from that given in most text-books. They show that, although pneumonia may be a comparatively mild disease in childhood, it is a severe and very fatal disease in infancy, the fatality varying inversely with the age of the infant. The duration of the fever in the 15 uncomplicated fatal cases in which it could be determined was 5, 7, 7, 9, 9, 10, 10, 11, 12, 12, 15, 15, 20, 22 and 28 days, giving an average of 12.8 days. (Average in cases which recovered, 8.1 days.)

In 23 of the fatal cases the pneumonia was uncomplicated. In the majority there was little or no resolution of the lung. Empyema was the cause of death in 4 cases, acute nephritis in 1, meningitis in 1, cardiac failure in 1 and thrombosis of the longitudinal sinus, with red softening of the brain, in another.

The following table shows which lobes were involved in the

fatal cases and whether they were involved in whole or in part. The percentages in parenthesis are the corresponding ones for the whole number of cases.

One Lobe.	Whole	Part	Total	Per Cent.
Right Upper Lobe -	6		6	26 (17%)
Right Lower Lobe -	1		1	4 (16%)
Left Lower Lobe - -	7	4	11	48 (38%)
	14=61% (52%)	4=17% (34%)	18=78%	(86%)

More than One Lobe.

Right Upper and Right Lower Lobes -	2
Three Right Lobes - - - - - - - -	1
Left Upper and Left Lower Lobes - -	2
	5=22% (14%)

This table shows that several lobes or the whole of a single lobe were somewhat more often involved in the fatal than in the combined fatal and non-fatal cases. This seems to justify to a certain extent the conclusion that the mortality varied directly with the amount of lung involved. The relative frequency with which the lobes were involved in the fatal cases was the same as in the combined cases, suggesting that there was no relation between the portion of the lung involved and the mortality.

The following table shows the total number of cases and the number of deaths for each degree of fever in the uncomplicated cases as well as the percentage of mortality for each degree both for the two years and for each year separately.

Highest Temperature.	First Year.		Second Year.		Totals.	
	Number of Cases.	Number of Fatal Cases and Per Cent. of Mortality.	Number of Cases	Number of Fatal Cases and Per Cent. of Mortality.	Number of Cases.	Number of Fatal Cases and Per Cent. of Mortality.
Over 106°F.	1	1—100 %	5	1—20 %	6	2—33 %
105°F -106°F.	16	5— 31 "	25	4—16 "	41	9—22 "
104°F.-105°F.	14	2— 14 "	13	1— 8 "	27	3—11 "
103°F.-104°F.	12	5— 42 "	16	2—13 "	28	7—25 "
103°F. or less.	5	1— 20 "	7	0—— "	12	1— 8 "
	48		66		114	22

This table shows that the mortality was lowest in the cases in which the temperature did not rise above 103°F., and highest in those in which it went over 106°F. The figures between these points are somewhat confusing. They seem to prove that a temperature between 104°F. and 105°F. was less serious than one between 103°F. and 104°F., or one between 105°F. and 106°F., both of which showed about the same mortality. This hardly seems reasonable, however, and it is probably safe to assume that variations in temperature between 103°F. and 106°F. had little effect on the mortality. A high temperature was apparently no more or no less fatal in the first than in the second year, the mortality in the younger infants being approximately twice as great for all temperatures in which there were cases enough to make comparisons of value.

The rates of the pulse and respiration in the fatal cases as well as the percentage of mortality for each rate, both for the two years combined and for each year separately, have already been shown in previous tables. As already noted, no case died in which the pulse was not over 140 or the respiration over 55. Differences in the pulse rate above 140, or in the respiratory rate above 55, had no apparent influence on the percentage of mortality, nor did there seem to be any definite relation between the rate of the pulse and respiration, the age and the percentage of mortality.

COMPLICATIONS.—*Empyema.*—There were 9 cases of empyema, giving a percentage of 7.6. Four of these occurred in the first year and five in the second, giving a percentage of 8 in the first and of 7.3 in the second year. While the percentage of cases of empyema was nearly the same in both years, empyema was far more fatal in the first than in the second year, 3 of the cases in the first year dying, and only one of those in the second, giving a mortality of 75 per cent. and 20 per cent. respectively. It was impossible to determine from the records when pus was first formed. The temperature, however, should be a fairly accurate guide. It did not fall in 4 cases, while it rose again the day after the crisis in 3 and three days after the crisis in 2.

Serous pleurisy occurred in 1 case, which was tapped. The fluid did not reaccumulate.

Otitis Media.—Otitis media occurred in 21 cases, giving a percentage of 17.8. It occurred equally frequently in both years. Both ears were involved in 14 cases, and one ear only in 7 cases. The process was catarrhal in 2 cases, and purulent in 19.

Cerebral Complications.—Thrombosis of the longitudinal sinus with extensive red softening of the brain occurred in 1 case. A pneumococcus meningitis, proved by lumbar puncture, developed in 1 case, which died. In 3 other cases there were marked symptoms of meningeal irritation. In one, death occurred in 12 hours. No lumbar puncture was made. In another in which there were local spasms and paralyses, followed by coma and death, two lumbar punctures gave negative results. In a third fatal case there was a severe terminal delirium lasting several days, the infant acting "as if insane."

Acute nephritis was the cause of death in 1 case. It developed early in the course of the disease and did not prove fatal until some days after the crisis.

Jaundice.—There was marked jaundice, accompanied by bile in the urine, in 1 case. The stools, however, were green.

TREATMENT.—No treatment beyond nursing and the regulation of diet was given in 30, or 25 per cent. of the cases. Antipneumococcic serum was used in 10 cases with no effect whatever on the course of the disease. Creosotal was used in 6 cases, also with no effect. Brandy was given in 70, or 60 per cent. of the cases, and strychnia in 30, or 25 per cent. of the cases. Aromatic spirits of ammonia was given in 12 cases, digitalis and caffein, each in 1 case. Phenacetin, in doses of ¾ grain to 2 grains, was used in 8 cases, and lactophenin in 2 grain doses in 1 case. The temperature was, as a rule, but little effected by these drugs. They apparently did no harm.

Cold in various forms was used in 28 cases, or 24 per cent. Tub baths, at temperatures varying from 80°F. to 100°F., were used in 8 cases. They were, as a rule, badly borne and in 2 cases caused serious collapse. They were, as a rule, not effectual. Sponge baths at from 80°F. to 90°F., or of equal parts of alcohol and water at 100°F., were used in 5 cases. They were usually resisted and fatigued the infants without giving any good results. Fan baths, at a temperature of from 100°F. to 110°F., were used in 14 cases. They were badly borne in 1 case, were disliked by another and caused excitement in a third. Several of the infants seemed to enjoy them, and, with the exceptions noted, they were always well borne. In most cases they caused drops in the temperature of from one to three or even more degrees. The cold pack was used in but 1 case, in which it gave very good results.

Cotton jackets and poultices were never used and no applications were made to the chest.

SUMMARY.—Lobar pneumonia is more common and occurs relatively more frequently in comparison with bronchopneumonia in infancy than is generally supposed. The analysis of these cases shows the following points:—The onset was less stormy than is generally thought. It was often ushered in by vomiting, but rarely by a convulsion. It usually began with fever and cough, which were often accompanied by apathy or drowsiness. Movement of the alæ nasi was not a constant symptom. A whole lobe was more often involved than a part. The left lower lobe was the one most commonly involved. The right upper lobe and right lower lobe came next in frequency. The portion of the lungs involved was relatively the same in the fatal cases as in those which recovered. As a rule, the area involved was larger in the fatal cases. The average duration of the fever in the cases that recovered was about eight days, being nearly the same in both years. The course was more often short in the first year, however, than in the second. The average duration of the fever in the uncomplicated fatal cases was 12.8 days; that is, the duration of the fever was longer in the fatal cases than in those that recovered. The highest temperature was usually between 103°F. and 106°F., the number of cases being nearly the same for each degree. The temperature fell by crisis in 68.8 per cent. Crises were less common in the first than in the second year. Collapse during the crisis was very unusual. Pseudo-crises were not very common, but irregularities and remissions in the temperature were not at all unusual. The mortality was lowest when the temperature did not rise above 103°F., and highest in those cases in which it went over 106°F. The degree of fever between 103°F. and 106°F. had no apparent effect on the mortality. A high temperature was no more fatal in the second year than in the first year. The usual pulse rate was between 150 and 170. No case died in which the pulse was not over 140. The rate of the pulse, when it was above 140, had little or no apparent influence on the mortality. The usual respiratory rate was between 55 and 80. No case died in which the respiration was below 55. The rate of the respiration, when above 55, had no apparent effect on the mortality. The mortality, excluding the cases in which death was due to empyema, was nearly 23 per cent. It was almost twice as great in the first as in the second year, being 32 per cent. and 18 per

cent. respectively. Otitis media was the most common complication, occurring in 18 per cent. Empyema was the next most frequent, occurring in about 8 per cent.

PROGNOSIS.—The following statements regarding prognosis seem justified. The prognosis of lobar pneumonia in infancy varies decidedly with the age of the infant and to a certain extent with the amount of lung involved. The prognosis is good when the temperature is not over 103°F. It is serious when it is above 106°F. Variations between these points make little or no difference in the prognosis. The prognosis is good when the pulse is not over 140 or the respiration over 55. The amount of the increase above these limits is of little importance.

DISCUSSION.

DR. KERLEY.—I should like to ask Dr. Morse if the respiration and pulse were taken when the child was asleep or when awake. During the past year the internes at the Infants' Asylum have made observations of interest and it was surprising how widely the respirations and pulse varied in perfectly normal children, the respirations being, of course, much slower when the child was asleep. More valuable information was obtained when these were taken while the child slept.

DR. ABT.—I would ask if any of the cases showed pronounced meningeal symptoms. We are led, from the literature, to believe that pneumonia with meningeal symptoms is relatively common.

DR. CHURCHILL.—The paper confirms my belief that lobar pneumonia is more common in children than has been supposed. I do not believe the cases are looked for carefully enough, or more would be discovered. I was much interested in a case of lobar pneumonia seen lately in a child of twelve months. The child was taken ill with pneumonia in February, there being only a small patch over the left lobe. Cultures from the throat revealed the presence of the pneumococcus and the smear showed influenza bacilli. The pneumonia subsided in thirty-six hours. Six weeks later the child was again taken ill with lobar pneumonia in exactly the same spot, ran a ten or twelve days' course and the child died. The culture showed the presence of the pneumococcus and no influenza bacillus was found. It was of interest that with a combined infection the affection was trivial; but when the pneumococcus infection existed alone it killed the child. The combination of pneumococcus and influenza infection in adults is almost invariably fatal. I do not understand whether or not pericarditis complicated the pneumonia in Dr. Morse's cases. I have

seen 2 such cases; in one there were no permanent after-effects; in the other a bad chronic endocarditis was left in its wake. In private practice the prognosis is much better than in hospital practice. I believe the giving of depressing antipyretics is more dangerous in infants than in adults. Sponge-baths are better borne than tubbing in children.

DR. HOLT.—The figures given by Dr. Morse are practically identical with my own statistics. The great frequency of involvement of the left lower lobe is still unexplained.

Regarding prognosis, it seems that pneumonia involving but a single lobe is seldom fatal, no matter how high the temperature might go, or how rapid the respiration might be, provided the child is in fair condition previous to the onset of the disease. Too much importance has been attached to the rapidity of the pulse. Certainly in a large proportion of the cases the pulse rate is over 150. It is the quality and not the frequency of the pulse which is the important factor. Cases may recover with an exceedingly high temperature even when it is let alone. I recall 1 case in which there was a temperature of 107.2°F., and yet the patient recovered. The antipyretic treatment of lobar pneumonia is a failure. It is a mistake to consider the temperature too much in treatment. Children, as a rule, bear antipyretic measures rather badly. I recall the instance of a boy, three and a half years old, with lobar pneumonia, whose father was a firm believer in antipyretic measures. When about to apply the cold sponging, the boy cried, "Papa, if you will let me alone I will never do it again."

The element of rest in the treatment of lobar pneumonia in children is very important. The ice-bag to the head is useful, especially for the nervous symptoms, which, when severe, call for the cold pack or even the bath.

The mortality differs in hospital and in private practice. Statistics collected some years ago gave a mortality considerably less than that given by Dr. Morse. I think it is a mistake to allow the opinion to go forth that the general mortality of lobar pneumonia is as high as has been given by Dr. Morse. With such a view, the inference would be drawn that the adoption of some particular plan of treatment is followed by remarkable success. If the text-books give a mortality of 30 per cent. for the first year, and the physician who treats all cases by large doses of iodid of potassium, sees results much better than this, it is not unnatural that he should claim that it is a specific for the disease. I do not believe the mortality from lobar pneumonia in children, taking all ages together, is over 10 per cent. in private practice.

DR. ADAMS.—I have been struck by the high mortality figures given by Dr. Morse since they do not accord with those in my own personal experience. My mortality was certainly not so high as given in the paper. Dr. Holt's figures of 10 per cent. would more nearly express my own.

I am glad that Dr. Holt called attention to the fact that the

thermometer is not such a great and important aid in the treatment of lobar pneumonia in children. The tendency is to regard high temperature as a menace of evil. I have frequently seen temperatures of 105°F. and 106°F. in children, without the slightest manifestations of nervous phenomena. I presume the temperatures given by Dr. Morse were taken per rectum.

Still, one is struck by the average low temperatures; the majority of the children did not run a temperature over 104°F. Certain children would bear a temperature of 104 or 105.5°F. without any manifestations of seriousness. I have long since abandoned the use of the bath except in extreme cases. One patient of mine had a temperature of 108.6°F.; clearing out the bowels and placing the child in the bath reduced the fever in two hours' time to 104°F.; but the left lung was solid from base to apex and the child was in a moribund state and died.

DR. NORTHRUP.—I prophesy that in two years a new element will be mentioned in the treatment of lobar pneumonia and that will be, *cool, fresh air,* which will be used as a heart stimulant and as an antipyretic and which will remove all nervous and cerebral symptoms.

DR. MORSE.—The pulse and respiration were taken when the infants were quiet, and if possible when asleep. I agree that the findings could not be relied upon unless the infants were quiet or sleeping. The point I wish to bring out in my paper is that there is no cause for alarm if the pulse and respiration are rapid.

With regard to the meningeal symptoms, there is no further history of these given than is recorded in the paper. Less careful observers would probably have thought that some of these infants had meningitis. Some of the cases had been sent in with the diagnosis of some abdominal lesion.

I did not enter into a discussion of the various plans of treatment, but have given only a summary of the treatment which had been followed in the hospital. I believe that it is very important to leave the patients alone as much as possible. They should have the greatest possible amount of fresh air, be properly fed and have the bowels kept open. If marked nervous symptoms develop as result of the high temperature, the temperature should be treated, otherwise, not. The most important part of the treatment is an abundance of fresh air. Personally, I have never given coal-tar antipyretics in pneumonia. My experience has led me to believe that infants do not bear tub-baths or sponge-baths as well as fan-baths. I feel that the prognosis in private practice is different from that in hospital practice. I do feel, however, that pneumonia in infancy is a much more serious disease than is usually supposed. Pneumonia in infancy is a very different disease from pneumonia in childhood. My experience with practitioners in general is that they think that babies with pneumonia almost always get well. I hope to make them realize by this paper that they do not always do so.

ACUTE OTITIS IN CHILDREN—A STUDY OF FIFTY-ONE OPERATIVE CASES IN PRIVATE PRACTICE.

BY CHARLES GILMORE KERLEY, M.D.,

Professor of Diseases of Children, New York Polyclinic Medical School and Hospital; Attending Physician, New York Infant Asylum; Assistant Attending Physician, Babies' Hospital, New York.

The 51 cases reported comprise the number of cases of acute otitis seen by me during the past two years. Six of these were seen in consultation. Nine had been previously treated by other physicians; they had been treated either for malaria, grippe or typhoid fever. Thirty-six occurred among my own patients. The ages of the cases treated were as follows:

3 months of age	2
5 " " "	2
6 " " "	3
8 " " "	1
9 " " "	3
10 " " "	2
12 " " "	3
13 " " "	5
14 " " "	3
15 " " "	1
16 " " "	1
20 " " "	2
24 " " "	3
30 " " "	2
3 years " "	2
4 " " "	4
5 " " "	2
6 " " "	4
8 " " "	4
10 " " "	1
14 " " "	1

7	of these	cases	were	between	3	and	6	months	of	age
9	"	"	"	"	8	"	12	"	"	"
10	"	"	"	"	13	"	16	"	"	"
7	"	"	"	"	20	"	30	"	"	"
12	"	"	"	"	3	"	6	years	"	"
6	"	"	"	"	8	"	14	"	"	"

In seventeen cases the patients were delicate—below the average in strength and development.

In thirty-four the patients were of normal development, and in previous good health.

In twenty-eight cases both ears were involved.

In twenty-three, one ear was involved.

In two, a second attack took place; one after three months, the other after five months.

The otitis was associated with or followed:—

German measles in	1	case
Scarlet fever in	2	cases
Measles in	7	"
Catarrhal colds in	38	"

The otitis was primary in three cases; in these there was no associated illness, no involvement of the respiratory tract.

Pus	was found in the middle ear in	48
Serum	" " " " " " "	3

Bacteriological examination showed:—

Streptococci in pure culture in	13
Staphylococci in pure culture in	11
Streptococci, staphylococci and pneumococci in	12
Staphylococci and pneumococci in	6
Staphylococci, pneumococci and colon bacilli in	1
Streptococci and staphylococci in	2
Pneumococci in	2
Bacteriological examination not made in	4

Pain and localized signs	were present in	17
" " " " "	absent "	34

In forty-four cases the otitis was uncomplicated.

In four there was mastoid involvement requiring operation.

In three there was jugular bulb involvement.

There was mastoid disease single in three cases; ages, 3 months, 9 months, and 8 years. There was mastoid disease double in one case, aged 9 months.

Bacteriological examination of pus in the mastoid cases showed:

Pure streptococci in..... 2
Streptococci and staphylococci in..... 2

All four cases made excellent recoveries; the wounds closing in from six to eight weeks.

In three cases there was thrombosis of the jugular bulb on one side. In two of these cases the mastoid structure was found normal upon operation. In one the mastoid was slightly involved. Examination of the pus from the ear in the bulb cases showed:—

Pure streptococci in..... 2
Pure staphylococci in..... 1

Dr. James F. McKernon, in an unpublished paper, says:

"The question may be asked: 'How can a primary involvement of the jugular bulb take place, without first involving the mastoid structure?' It can take place very easily under the following conditions: With an active, virulent infection of the middle ear cavity, and the presence of a large, irregularly shaped bulb encroaching upon it, and these two spaces separated by only a thin, parchment-like wall of bone, it can become a very easy matter for the infection to pass through this thin plate of bone and poison the blood current, before such infection would have time to travel its usual pathway, namely: through the mastoid structure. That such a complication is not infrequent can be readily comprehended when, in the writer's experience alone, seven such cases have been encountered during the past six months. The successful treatment of this condition is an early operative procedure."

In thirty-three of the uncomplicated cases the discharge ceased and the drum closed in from thirteen to fourteen days, and in seven cases from fourteen to twenty-one days. In four out-of-town cases the exact duration could not be learned. The treatment employed after paracentesis was syringing with eight ounces of 1-10,000 bichlorid every three hours for twenty-four hours, or every four hours for twenty-four or forty-eight hours, the frequency of the syringing and the number of days it was con-

tinued being dependent upon the nature and amount of the discharge.

Interesting points brought out by the study of the cases are as follows:

The physical condition of the child appeared to exert no influence upon the susceptibility; two-thirds of the patients were strong, well-nourished children. In none were the tonsils sufficiently involved to require removal. Six cases had sufficient nasal obstruction to suggest the presence of adenoids—they had chronic nasal discharge; were mouth-breathers; and had a persistent cough.

The fact that thirty-eight of the cases, or seventy-two per cent. developed with or followed catarrhal inflammation of the upper respiratory tract, socalled catarrhal cold, emphasizes the necessity for frequent aural examination during and following such disorders, particularly when there is an elevation of the temperature —the temperature which, in the absence of definite clinical signs, we are apt to look upon as due to chronic grippe, malaria, paratyphoid, dentition, and so forth.

The most interesting clinical feature of the study of the cases was the absence of pain or local signs of disease in thirty-four, or sixty-seven per cent. Among those tabulated in the pain group there are a few which perhaps should not be so recorded. In these there were no manifestations of pain such as we generally expect to find, as evidenced by crying, pulling at the ears, or striking at the sides of the head. Neither were there signs of pain or unusual discomfort on local manipulations. Among the pain group are included those who were restless, who slept poorly, or who appeared in any way uncomfortable. Had it been left to pain or local symptoms, a diagnosis of otitis, in thirty-four of the cases, would not have been made at the time it was made. The ears in these cases were examined because of the presence of fever, which could not be accounted for by any of the means which may produce an elevation of the temperature in children.

In the six consultation cases, and in the nine cases which had been treated by other physicians, the possibility of ear involvement had not been considered because of the absence of pain or localized signs.

The diagnosis of otitis was confirmed in each case by well-known New York aurists.

Six of the mastoid and jugular bulb cases followed catarrhal

colds. One followed measles. As to the nature of the infection in the ear, bacteriological examination showed the presence of streptococci in pure culture, or associated with staphylococci in all but one case. In this one there was pure staphylococcus infection. In these complicated cases there was never pain, as shown by crying or restlessness; nor was any sign of discomfort elicited by manipulation. Neither was there evidence of involvement of the deeper bony parts through swelling or deformity.

In one only, the seventh case, a girl eight years of age, were there local signs of mastoid disease. This child showed a moderate amount of postauricular swelling, and a displacement forward of the ear.

In three of the mastoid cases the diagnosis was made by the continuation of the temperature after the incision of the drum membrane, a prolapse of the posterior-superior wall of the canal, and the absence of all other clinical manifestations of an explanatory nature. In mastoid disease in adults and older children the temperature only exists during the onset of the cellular infiltration —when the pus has once formed the temperature remains low until such time as there is involvement of the adjacent structures. In infants and young children the temperature is one of the most valuable aids, and is, it can safely be said, always present when there is confined pus in the mastoid. In the cases with bulb involvement the diagnosis was made by the rapid rise and fall of the temperature, following upon acute streptococcus infection of the middle ear, and without other assignable cause for the temperature.

CHART I. The case of which Chart I. shows the temperature range was a girl two and one-half years of age, in previous excellent health. The case began as an acute catarrhal cold, with congestion of the fauces, moderate swelling and congestion of the tonsils. Under treatment the child rapidly improved, the throat being normal on the third day of illness. In spite of this the fever continued, with no evidence of pain or discomfort of any nature whatsoever. The ears were examined with a view to explaining the temperature. Both drums were found bulging. Paracentesis was performed by Dr. McKernon on the fifth day of illness. There was pus in both ears. On the next day, the sixth day of illness, the temperature rose from 100° to 105½° in three hours. At the same time there was a free discharge of pus from both ears. On the

following day there was a fall to 99°F., and a rise not quite as sudden to 105°. On the seventh day the temperature ranged between 100° and 102°; and on the eighth day there was another rapid rise from 100° to 105°. There was no pain, and no mastoid tenderness. The child was comfortable and playful when the temperature was low; irritable, restless and drowsy when the temperature was high. Preceding and during the rise, the hands and feet were cold. On the eighth day the child was seen in consultation by Drs. Dench, A. A. Smith and McKernon. No cause for the fever could be demonstrated by physical examination, by examination of the blood, or of the discharge from the kidneys or bowels. Examination of the pus from the ears showed pure

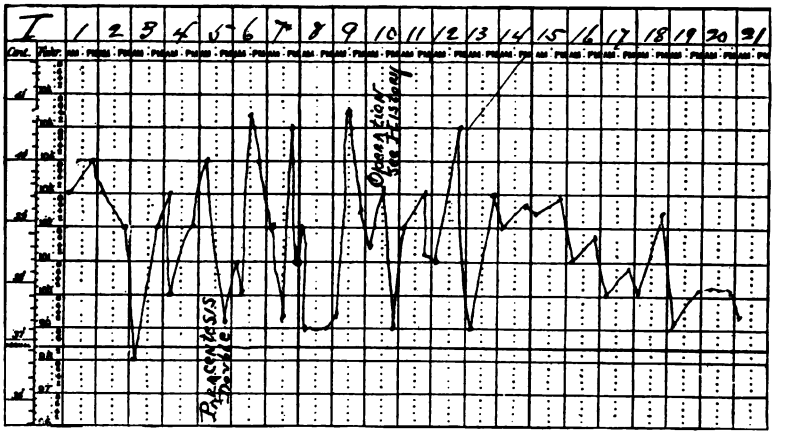

CHART I.

streptococci. The diagnosis of jugular bulb involvement of the left side was made. The next day, the tenth day of the illness, Dr. McKernon opened the mastoid and exposed the sinus from the knee to the bulb. The mastoid was found normal, the sinus was empty. There was a clot in the jugular bulb one inch long, extending a quarter of an inch into the sinus. The clot was examined by Dr. Frederick Sondern, who reported that it contained numerous foci of pus. Cultures made from the same material on nutrient agar and on Löffler's blood serum showed, after eighteen hours at 37°C., a decided growth of streptococcus pyogenes. The child made an uneventful recovery; the wound closing in six weeks.

CHART J. The second case, the temperature of which will be

seen on Chart J, was a boy, three years of age, in good average health. He had been ill two days with a catarrhal cold which was not sufficiently severe to produce a temperature of 104° to 105°. The ears were examined on the second day, with a view to explaining the high temperature. There was no pain, no tenderness about the ears, no symptoms other than the temperature to attract attention to the ears. The right drum was found bulging. A paracentesis was made by Dr. McKernon. The following day the temperature arose to 104°, when the left drum was incised. There was a free pus discharge from both ears; and a sudden drop in the temperature, was followed by a rise the next morning to 105°, the

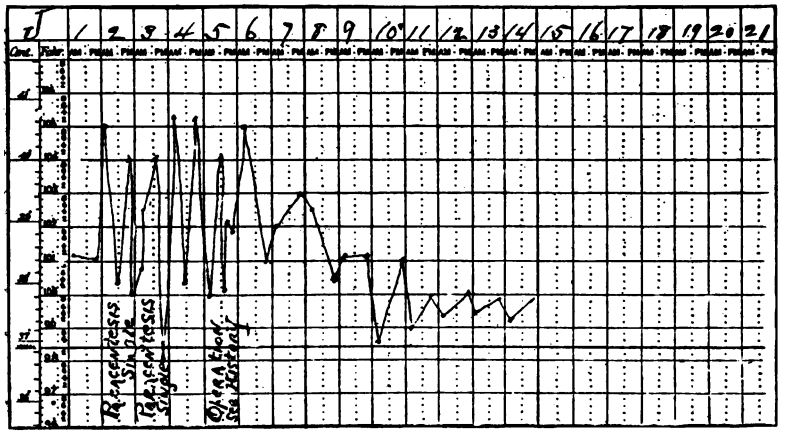

CHART J.

rise taking place in six hours. During the next thirty-six hours there were wide fluctuations. Physical examination was negative. Blood examination for Widal and for plasmodia negative. On the fifth day of the illness Drs. McKernon and Gruening saw the case in consultation. A diagnosis of jugular bulb infection was made and immediate operation advised. The operation was performed by Drs. McKernon and Gruening. The mastoid structure was normal. A clot was found in the right jugular bulb, completely blocking the sinus. The bony wall covering the jugular bulb was entirely removed. The child made an excellent recovery, the wound closing in six weeks. Bacteriological examination of the original infection in ear showed pure streptococci. The bacteriological examination of the clot by Dr. Frederick

Soudern showed pure streptococci on culture. Streptococci were also found in the blood on culture.

CHART H. The third sinus case was that of a girl 13 months of age. The child developed measles, from which she recovered; the temperature having been normal for three days. The next day, the first day indicated in the Chart, it ranged between 99° and 100°; and the following day it rose to 104°. The child was restless and without appetite. Physical examination was negative with the exception of both ear drums, which were found bulging. There was no evidence of pain, no tenderness about the ears. Paracentesis was performed by Dr. McKernon, followed by a free discharge of pus without influencing the

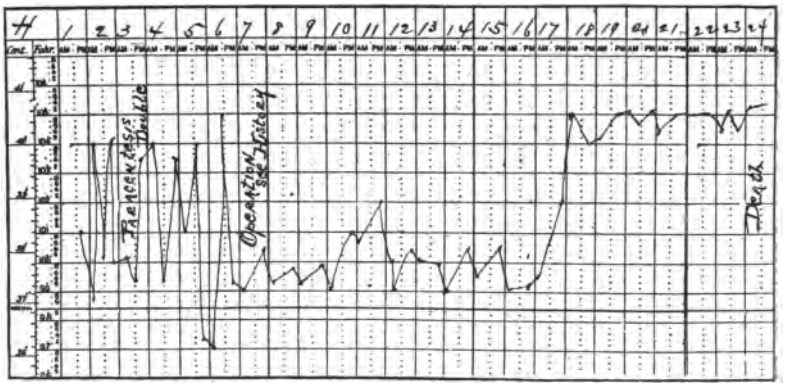

CHART H.

temperature, which ranged between 99° and 104° for the next five days. There was no mastoid tenderness, and no swelling. The child at this time, the sixth day of the illness, was seen in consultation by Drs. McKernon, Gruening and Holt, the diagnosis of jugular bulb infection made, and operation decided upon. The operation was performed by Dr. McKernon, assisted by Dr. Mason. The mastoid structure on the right side was found diseased. The sinus was exposed and a clot removed which was extended into the jugular bulb. The child did well for ten days. Then the temperature arose and ranged from 103° to 105° for eight days, when she died of encephalitis. Bacteriological examination by Dr. Frederick Soudern of the clot found in the jugular bulb showed pure staphylococcus on culture.

CHART K. Chart K shows the temperature range in a delicate,

anemic child one year of age. The parents gave a history of illness of two weeks, with cough and fever. She was under treatment for grippe; and the temperature which had been taken had ranged from 100° to 103°. Physical examination and examination of the blood failed to show a cause for the continued fever. There had been no earache, there was no local tenderness at the time when I saw the child. The ears were examined with a view to explaining the temperature. Aural examination showed bulging of the drum on the right side. Paracentesis was performed by Dr. McKernon. Three days later the left drum was incised. After this the temperature was lower, ranging between 99° and 102° for six days, with loss of flesh and strength. Dr. Gruening and Dr. McKernon

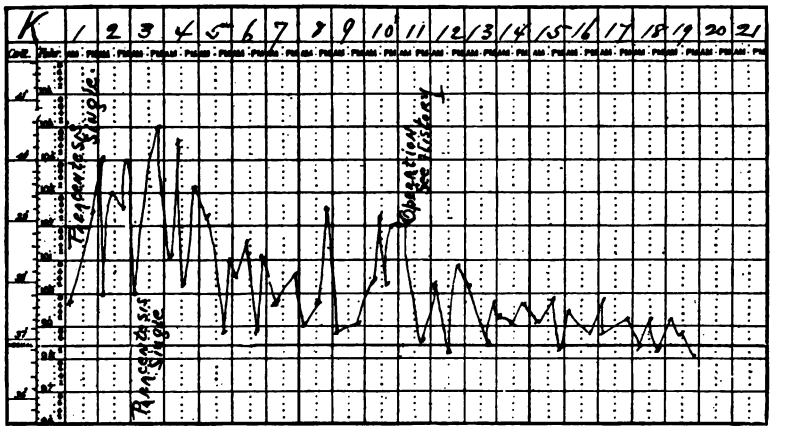

CHART K.

then saw the case in consultation. There was no pain, no mastoid tenderness, no swelling. Otoscopy showed prolapse of the posterior-superior canal wall and a granular point at the lower portion of the incision. A diagnosis of mastoiditis on the right side was made. Operation on the mastoid was performed by Dr. McKernon, assisted by Dr. Gruening. The mastoid cells were broken down and filled with pus. Bacteriological examination showed staphylococci, with a few streptococci. The drum closed in six days, and the mastoid wound in eight weeks.

CHART L. Chart L shows a temperature range of acute otitis, mastoiditis and adentitis, in a delicate child three months of age, who had just recovered from an acute attack of double bronchial pneumonia. The child developed an acute catarrhal

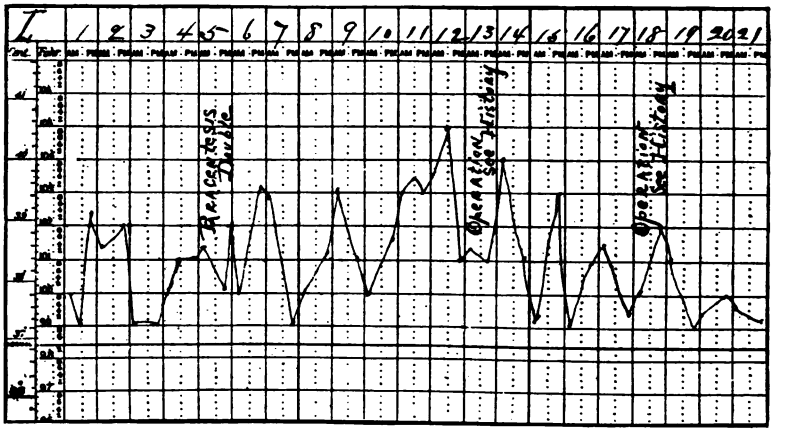

CHART L.

cold, which was readily controlled in two days. The temperature was normal on the third day of the illness. It arose to 101° on the fourth day, and to 102° on the fifth day. In the absence of other clinical signs, the ears were examined to explain the cause of the fever, both were found involved, and incision of the drum membrane was made by Dr. McKernon, followed by a free discharge of pus. The temperature was not influenced in any way, it remaining very irregular—touching 99°, and going as high as 105° during the following eight days. There had been no evidence of pain, no swelling, no tenderness. Bacteriological examination of the pus from the ears showed pure streptococcus infection. A considerable adenitis developed on both sides. On a continuation of the temperature, absence of physical signs to explain the temperature, and prolapse of the posterior-superior canal wall, mastoid disease was diagnosed; and operation for mastoiditis performed by Drs. McKernon and Mason. The right mastoid was opened and found filled with pus. Three lymphatic glands at the angle of the jaw, on the same side, were removed five days later. The largest of the three was found broken down in the middle; the bacteriological examination of which showed pure streptococci. The parts were slow of healing, on account of the weakened condition of the child. The wound closed in eight weeks, and the child recovered.

CASE III., MASTOID. The temperature chart for the case of mastoid in the eight-year-old girl is not shown, for the reason that

there was no temperature. For two or three days there had been slight pain in and behind the ear; and a day or two of slight discharge, which was further noticed when the case was first seen. There was also moderate swelling behind the ear, pushing the ear forward. The child had suffered with earache during the previous night. The drum was found bulging. Operation for mastoiditis was performed by Dr. McKernon. Examination for pus showed streptococci. The wound closed in four weeks.

CASE IV., MASTOID. The temperature chart of the fourth case of mastoid disease was taken away by the nurse and could not be recovered. The patient, nine months old, had been ill for two weeks, in the care of another physician, with rather indefinite symptoms of fever, restlessness, and loss of appetite. The temperature had been taken but a few times; and it had never been above 103°. In a routine examination the ears were included and both drums found bulging. Otherwise there was no evidence of disease. Paracentesis revealed pus in both ears. The child did not improve after the operation, and the fever continued as before. Six days after the paracentesis a diagnosis of mastoid disease was made, the mastoid cells were opened on both sides, and pus was found. There was no mastoid tenderness on either side. Bacteriological examination of pus revealed streptococci and staphylococci. The child made a good recovery.

CHART A. Chart A shows the temperature range in a case of

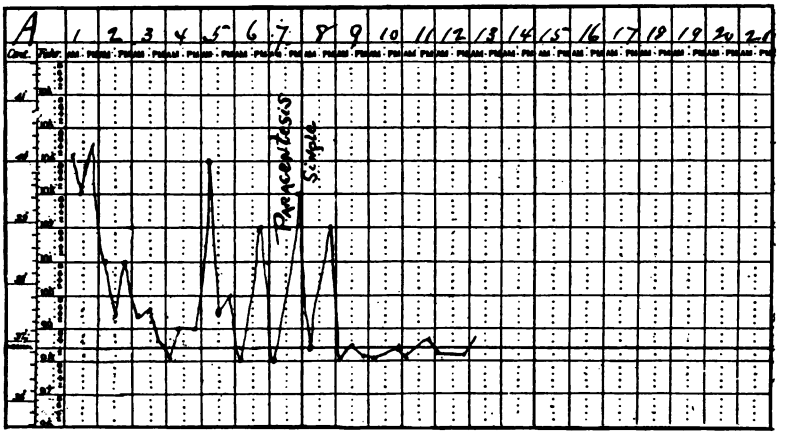

CHART A.

acute otitis following German measles in a child five years of age. The first day the temperature was high—104½°—with the appearance of a well-marked rash. The fever subsided on the second day, and remained normal the following two days. On the fifth day of the illness the temperature arose to 103.8° without explainable cause, and dropped without interference to 99.5°. The next day, the sixth day of the illness, it arose to 102°, and in the morning it dropped to normal. On the seventh day the temperature arose to 103°. There was no pain, nothing to attract attention to the ears. They were examined with a view to locating the cause for the temperature. The right drum was found bulging. Paracentesis was performed by Dr. McKernon, and serum found. Bacteriological examination showed a mixed infection: streptococci, staphylococci and pneumococci.

CHART B. Chart B shows the temperature range in a case of acute double otitis. The patient was a strong, vigorous boy fourteen months of age, who was taken ill with high fever and a

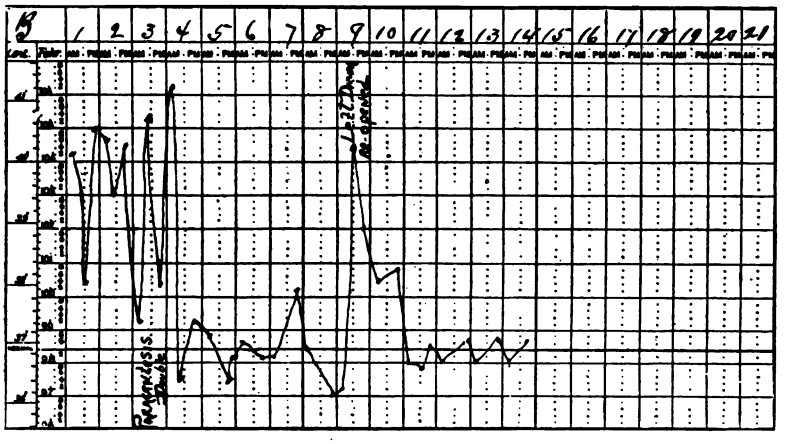

CHART B.

slight redness of the fauces. The respiration was from 50 to 60. The chest was clear; but as a cause for the rapid respiration a diagnosis of a probable central pneumonia was made. On the third day of the illness I saw the patient when the temperature was low. At this time the respirations were 24. The child was playful, and appeared to be in no way inconvenienced. There was not, nor had there been, any evidence of pain in the ears; and there was no localized tenderness about the ears. The blood was negative for

plasmodia. The ears were examined to explain, if possible, the temperature. Both drums were found bulging. Paracentesis was performed by Dr. McKernon, who found pus in both ears. On the ninth day of the illness the temperature arose to 104.5° F. It was found that the left drum had closed; this was reopened. The child made an uneventful recovery. Bacteriological examination showed pure streptococci.

CHART D. Chart D shows the temperature range in a case of acute otitis in a rather delicate boy, six years of age. He had been ill a couple of days with a catarrhal cold, the temperature not going above normal. On the third day the temperature arose

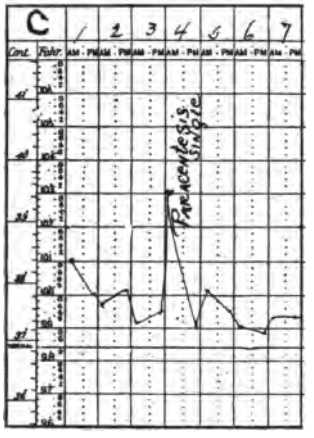

CHART C.

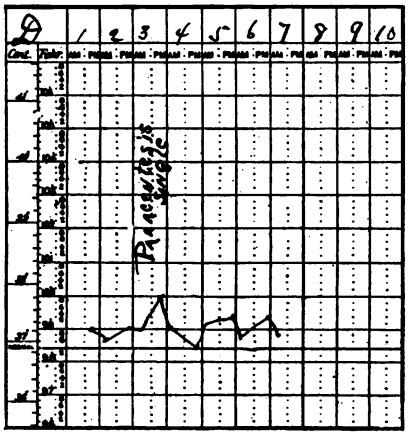

CHART D.

to 100°. During this day the child suffered from excruciating earache. Among all of the fifty-one cases recorded this boy suffered the most pain. Examination of his ear showed a red and bulging drum on the left side. Paracentesis was performed by Dr. Gruening. A small amount of serum was found. There was no further pain, and the child was well in seven days. Bacteriological examination of the serum was not made.

CHART E. Chart E shows the temperature range in a case of acute otitis in a child thirteen months of age. The patient was pale and thin; there was a history of several weeks of low fever, during which time she had been under the care of a physician. During the week previous to my seeing the case, which dates from the second day of the chart, the temperature had been ranging

rather high—from 101° to 104°, according to the mother; but it had not been taken regularly. The day previous to my visit it had been as high as 105.3°. At the time of my first visit it was 101°. The following day it remained 100°. The next morning it arose to 103°; and remained between 103° and 105° all day. Inability to find an explanation of the temperature led to an examination of the ears, and both drums were found bulging. Paracentesis was performed by Dr. McKernon. A large amount of pus was present in the right ear, a small amount in the left. The temperature promptly fell to the normal after the paracentesis, and remained there, except for a rise a few days after, which was due to over-feeding. Bacterio-

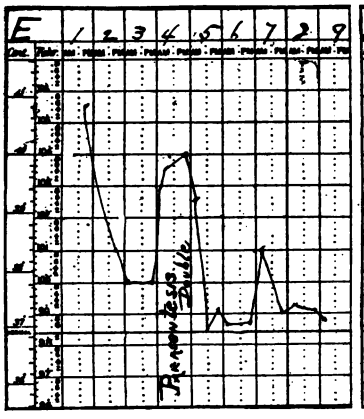

CHART E.

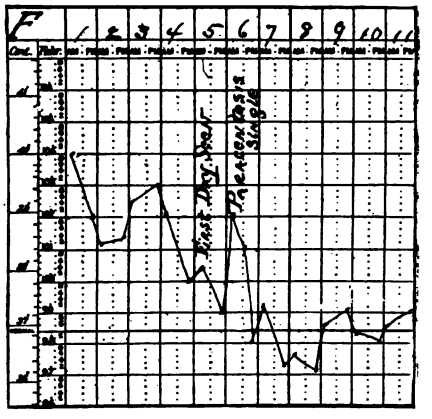

CHART F.

logical examination of pus showed streptococci, and a few diplococci.

CHART F. Chart F shows the temperature range in a delicate nine months' old girl. The mother gave a history of an illness of six weeks' duration, in which the persistent temperature, which up to this time had not been explained, had been the most prominent symptom. She informed me that I was the eighth physician who had seen the case. Blood examination had been repeatedly made for typhoid and malaria. The child was being treated for malaria when I was called. Physical examination was negative, with the exception of the right ear drum, which was found bulging. Accurate temperature observations were obtainable only for four days before my visit. There was no pain, no localized

tenderness, not the slightest evidence that the ear was involved. Paracentesis was performed by Dr. McKernon. Bacteriological examination of the pus showed pure streptococci. The child made an excellent recovery, with no further rise in temperature; and the wound closed on the tenth day after the paracentesis.

CHART G. Chart G shows the temperature range in a strong, vigorous child, twelve months of age, who had been ill one day, with a slight catarrhal cold. There was very slight congestion of the fauces. The temperature was 102° when first seen, but it dropped to normal early in the day, and remained at 99° or below the next day. The child was discharged well on the second day. The following day the temperature arose to 102°, and fell to normal the next

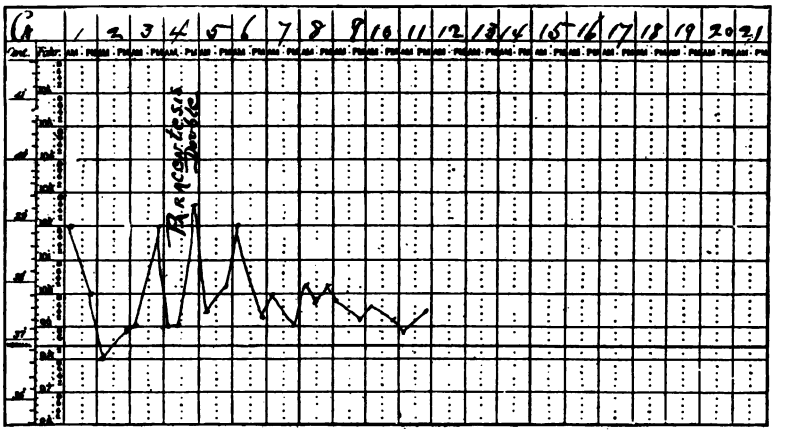

CHART G.

day. In the evening of this day, the fourth day of the illness, there was a sudden rise to 102.8. In looking for the cause the ears were examined, and the left drum was found bulging. There was no pain, no discomfort whatever. Paracentesis was performed by Dr. McKernon, and a free discharge of pus followed. Bacteriological examination showed pneumococci predominating, with a few staphylococci. The drum closed and the discharge ceased in eight days.

CHART C, on page 755, shows the temperature range of a strong, vigorous boy, three years of age. There was a history of slight fever, the highest temperature 101°, without assignable cause on the part of the family. I was called to see him because of a sudden rise of the temperature to 103°, which is recorded on the chart

as having taken place on the fourth day. In examination to discover the cause, the ears were examined last. The left drum was found bulging. Paracentesis and a free discharge of pus was followed by prompt recovery. Bacteriological examination showed streptococci.

In conclusion, I would call attention to a very interesting condition which was present in each of the mastoid and sinus cases. The children did not appear very ill. They felt and acted as though they were ill only when the temperature was high. When the temperature was low, they sat up, played, and were fairly bright. It was difficult in every instance to make the parents realize the necessity for operative procedure. Had not the source of the infection been early discovered, the children would, of course, have soon showed the usual signs of pyaemia.

When practitioners realize the possibilities for serious troubles that the middle ear affords, when they become more familiar with otoscopy, there will be fewer cases of meningitis, few cases diagnosed as typhoid independent of the Widal reaction, fewer cases diagnosed as malaria in the absence of plasmodium in the blood, fewer chronic grippes and fewer paratyphoids.

DISCUSSION.

DR. ROTCH.—On account of the latency of the aural symptoms, the general practitioner does not, as a rule, recognize these cases of otitis media. Pneumonia is another disease which should be spoken of in this connection. I have seen cases, occurring in babies and young children, in which the symptoms apparently were those of a central pneumonia, with the alae nasi working, and other symptoms; the respiratory symptoms were relieved by treating the ear. In cases in which the respiratory symptoms were relieved by inflating the Eustachian tubes and relieving the congestion of the ear this relationship was shown. Cases of otitis media with a discharge from both ears, unless we are skilled in the examination of the ear, should be referred to an aurist. In childhood there is much more danger of infection of the meninges than in adults. I would insist upon the importance of a free incision in the tympanum in order to prevent such complications.

DR. KERLEY.—One of the cases reported I diagnosed as central pneumonia. I happened to see the child always when the temperature was high—105° to 106° F. The child was one year old and the number of respirations per minute was 60 to 75. On the third day

of the illness I saw the patient when the temperature was 99.5 and the respirations 24. An examination of the ears revealed the cause of the fever. I had not examined the ears before because I was sure of the diagnosis of central pneumonia.

DR. ROTCH.—I remember two children who impressed me with certain points. One was a baby seen during the period of dentition. The baby was taken sick in the evening with a temperature of 103°F. which was rising, with rapid respirations, the alae nasi working rapidly and the diagnosis of pneumonia was made. By inflating the Eustachian tubes and relieving the congestion of the middle ears the symptoms disappeared. The second case was that of a child whose temperature rose to 104°F.; the child was sleeping quietly and the respirations were normal. A pneumonia was evidenced by percussion and auscultation.

DR. MORSE.—I presented a paper on this same subject at the American Medical Association last year. My experience has led me to believe that disease of the ears is very common in babies and in young children, and that in most of the cases the true condition is not recognized and that the symptoms are attributed to some other cause. Most often, of course, it complicates some disease of the respiratory tract, especially simple cold, but it often occurs in the course of other diseases and is not infrequently primary. In a considerable number of cases the symptoms are latent or are disguised by those of the primary disease. I feel sure that at least one-third of the patients in the Infants' Hospital always, and sometimes two-thirds of them, have inflammation of the ears. Inflammation of the ears is especially common as a complication of pneumonia. It should not be forgotten that a pneumonia and disease of the ear may exist at the same time. I feel very strongly that the frequency of diseases of the ears is not recognized by the profession at large. I think the reason that they were not recognized is because definite symptoms are looked for. Physicians, as a rule, exclude ear trouble without further examination if there is no earache, tenderness over the mastoid or picking at the ears. Earache, however, is very often not present, or if present, the crying is misinterpreted. Tenderness over the mastoid is unusual in acute middle ear disease. Babies will cry anyway if pressure is made over the mastoid. Babies very seldom put their hands to their ears or head when there is disease in the ear and very often do it in other conditions. The only way that a diagnosis of middle ear disease can be made is by the ear speculum.

A CASE OF CARBOLIC ACID POISONING SIMULATING ACUTE SCARLATINAL NEPHRITIS.

BY SAMUEL S. ADAMS, A.M., M.D.,

Washington, D. C.

Q. R., aged ten years, female, white, was visited February 3, 1904. The child had a moderately severe attack of scarlet fever without any troublesome symptoms. I saw her daily until February 9th, and thereafter on the following dates: 11th, 14th, 17th and 19th. She was nursed by her mother who was constantly insisting that the symptoms being so slight there was no necessity to keep the child in bed. I insisted, however, upon the instructions as to the general management being strictly carried out. It was impossible from beginning to end even to approximate the quantity of urine passed in any twenty-four hours, as the mother invariably had an excuse for not keeping it or not measuring it.

Urine Analysis No. 1.—February 5th.—Dark amber; cloudy; acid; 1.029; albumin, 0; sugar, 0; urea, grs. xiii to ℥i. Sediment shows large number of crystals of calcium oxalate; many desquamated cells, mostly squamous epithelium; few degenerated leukocytes. This and the succeeding urinalyses were made by Dr. Thomas S. Grasty.

At my visit on the 9th, the child complained of intense itching, so I instructed her mother to bathe her twice daily with a teaspoonful of carbolic acid to the quart of water (1-256). She handed me a 12 ounce bottle, which she said contained 1 ounce of carbolic acid to 11 ounces of water and asked how she could use it. I then told her to take a tablespoonful of her solution to the quart of warm water for the bathing (1-800).

I visited the patient several times between the 9th and 16th. Another examination of the urine was made as follows:—

Urine Analysis No. 2.—February 16th.—Pale amber; cloudy; faintly acid; 1.021; albumin, 0; sugar, 0; urea, grs. iv. to ℥i. Microscope shows urine loaded with crystals of calcium oxalate; few cells seen.

She was visited again on the 19th and found to be in excel-

lent condition. Desquamation was very profuse and she was being judiciously fed. Her condition was so good I ordered chicken to be added to the diet. The itching had been allayed by the baths, there was neither tingling nor irritation of the skin from the carbolic acid and she was ordered to be kept in bed as long as desquamation continued.

On the morning of February 22d, I received a note from the mother telling me that the child was bright and happy, but had passed a fair quantity of dark urine, a specimen of which she sent to me.

Urine Analysis No. 3.—February 22d.—Smoky; cloudy; acid; 1.031; albumin present; sugar absent; urea, grs. ix to ℥i. Sediment shows red blood cells, hyaline and granular casts all in only moderate numbers; epithelial and pus cells; few uric acid crystals.

I saw the patient, whose eyes were only slightly puffed, otherwise she was in fine condition. Even now it was impossible to form any accurate idea about the amount of urine passed, but I satisfied myself that there was only a slight diminution of the daily excretion. Baths were stopped. The following day I had a specimen of urine examined at the Children's Hospital, which showed the absence of albumin and sugar, specific gravity, 1.038; urea, 12 grains to the ounce; a large number of red blood cells and a few cylindrical epithelial cells. The various tests for carbolic acid proved that the urine contained it.

February 24th. The puffiness has disappeared and she has no complaint.

Urine Analysis No. 4.—February 24th.—Smoky; cloudy; acid; 1.020; albumin, present; sugar, absent; urea, grs. vii to ℥i. Sediment shows few hyaline and a moderate number of coarse and finely granular casts; few blood cells and pus cells; epithelial cells; no crystals.

Urine Analysis No. 5.—February 26th.—Amber; very slightly dirty in color; slightly cloudy; very faintly acid; 1.023; albumin, slight trace present; sugar, absent; urea, grs. viss to ℥i. Sediment shows moderate number of granular casts; pus cells; few, epithelial cells; an occasional hyaline cast.

Urine Analysis No. 6.—February 29th.—Pale straw color; cloudy; faintly acid; 1.018; albumin, slight trace present; sugar, 0; urea, grs. vss to ℥i. Centrifuge shows hyaline and granular casts, both fine and coarse, in moderate numbers; pus and epithelial cells; no blood.

March 5th. She has steadily improved and the urinalysis showed:

Urine Analysis No. 7.—March 5th.—Pale straw color; cloudy, flocculent precipitate; acid; 1.030; albumin, 0; sugar, 0; urea, grs. viii to ℥i. Centrifuge shows an occasional granular cast; moderate number of pus cells; epithelial cells; much dirt and many bacteria.

March 9th. I discharged the patient, but warned her parents to notify me if any further trouble arose. She was now passing a large quantity of urine.

Urine Analysis No. 8.—March 9th.—Pale amber; cloudy; distinctly acid; 1.028; albumin, trace present; sugar, 0; urea, grs. vii to ℥i. Centrifuge shows a few casts, hyaline and granular; pus cells; few epithelial cells; uric acid crystals (few).

May 9th. I found the child in first class condition; in fact, far above her average health, weight and spirits. Urinalysis showed:

Urine Analysis No. 9.—Pale; cloudy; acid; 1.025; albumin, 0; sugar, 0; urea, grs. v to ℥i. Centrifuge showed amorphous urates; pus cells (few); epithelial cells (squamous).

Having previously reported cases of carbolic acid poisoning following its use on the skin, I was satisfied from the urinalysis made on February 22d that the nephritic symptoms were due to the irritation of the drug and not to the toxemic effect of the scarlatina. This opinion was confirmed by the speedy relief of the kidney irritation *pari pasu* with the elimination of the carbolic acid. Such a weak solution of the acid was used it seems reasonable to attribute the toxic effects to an idiosyncrasy of the patient.

A CURE OF CHRONIC NEPHRITIS FOLLOWING RENAL DECAPSULATION.

Second Report.

BY AUGUSTUS CAILLÉ, M.D.,
New York.

The patient is a girl seven years of age, who was four and one-half years old at the time of operation.

History prior to operation. At age of two years contracted measles. Two weeks later fell into a tub of scalding water, and received very severe burns of back and legs. About two weeks after that swelling of feet and puffiness of face appeared, followed a little later by ascites. "A well-known physician diagnosed the case as one of acute nephritis, from which she apparently recovered after four weeks. A similar attack, a month later, also ended in apparent recovery. On the occasion of a third attack, which was more severe, with supression of urine of forty-five hours' duration, the child was admitted to the Babies' Wards of the N. Y. Post Graduate Hospital, November 13, 1900. Her case was diagnosticated as subacute nephritis and she was discharged, improved, December 30, 1900. On April 3, 1901, she was readmitted during my service, with all clinical evidence of chronic nephritis; urine scanty and dark, containing albumin, all forms of casts and renal elements, blood and pus. Her eyes were puffy and her abdomen contained fluid. After a course of sweating, dieting, the application of intestinal irrigation, and the administration of diuretics and diaphoretics, she went home improved. Nine months later, on February 1, 1902, I found her again an inmate of the Babies' Wards."

Examination. "She was generally edematous, her heart was markedly enlarged, the apex beat being at the sixth interspace, an inch to the left of the nipple line. The heart sounds were clear, the second sound was accentuated."

Existence of chronic Bright's disease prior to operation. (a) as indicated by symptoms, two and a half years. (b) as known from urine examination, two and a half years.

Operation, February 19, 1902, at Post Graduate Hospital. Child four and one-half years old.

Decapsulation of both kidneys by Dr. G. M. Edebohls.

Anesthetic. Nitrous oxid and ether.

CASE NO. 21. C. B. WEIGHT OF PATIENT, 23 KILOGRAMS.

Operation Feb. 19, 1902.	Quantity of urine in 24 hours.	Specific Gravity.	Total solids in 24 hours.	Urea.	Albumin.	Renal Casts.
Eight examinations before operation, Feb. 6–13, 1902.	495 c.c. to 840 c c. Average, 700 c.c.	1010 to 1022. Average, 1017.	Average for 8 days, 27.0 grams daily.	Average for 8 days, 8.5 grams daily.	Average for 8 days, 0.103% daily.	Hyaline casts always. Granular and epitheliated generally. Few pus cells generally, and occasionally a red blood cell and renal element.
Eight examinations immediately after operation, Feb. 21–28, 1902.	330 c.c to 750 c.c. Average, 540 c.c.	1012 to 1029. Average, 1018.	Average for 8 days, 21.56 grams daily.	Average for 8 days, 9.288 grams daily.	Average for 8 days, 0.1% daily.	Same casts as before, but for first few days much more abundant. Also waxy, pus and occasional epitheliated casts. Blood and pus cells about same as before. Occasional renal cells.
Eight examinations made between Mar. 1–22, 1902.	510 c.c. to 1065 c.c. Average, 750 c.c.	1009 to 1032. Average, 1021.	Average for 8 days, 36.72 grams daily.	Average for 8 days, 18.3 grams daily.	Average for 8 days, 0.078% daily.	Large numbers granular casts, fewer hyaline. Nearly every report shows a few renal cells and pus cells.
May 20, 1902.	660 c.c.	1027	41.52 grams.	21.78 grams.	0.075%	Few leukocytes and renal epithelia.
Feb. 4, 1903.	1000 c.c	1012	27.76 grams.	18.0 grams.	0.05%	No casts, no red and no white blood cells. Five to six small epithelial (renal?) cells.
Aug. 11, 1903.	1000 c.c.	1020	46.6 grams.	14.0 grams.	Trace.	Rather frequent hyaline. Rarely an epitheliated hyaline. Rarely a red cell; occasional leukocyte.
Jan. 4, 1904.	1000 c.c.	1019	44.27 grams.	24.0 grams.	Faint trace.	Very rarely a hyaline. Very rarely a red cell. Few leukocytes.

Condition of kidneys at operation. Typical chronic parenchymatous nephritis, the large white kidney, on both sides. "Each kidney measured over four inches (ten centimeters) in length and was three times larger in bulk than normal."

Diagnosis. Right and left chronic parenchymatous nephritis.

Convalescence uneventful. Primary union of both wounds.

Urine examinations were made in large number, at least thirty. In the subjoined table I have averaged eight urine exami-

nations before operation, eight immediately after operation and eight made between ten and thirty days after operation. In addition, I have given all the urine examinations I have been able to make since patient left hospital. The comparison of the various examinations is certainly most instructive.

Examination of urine by Arthur H. Gardner, M.D., April 16, 1904. Urine of C. B., seven years old.

Color—very pale straw.

Specific gravity 1021.

No albumin.

No sugar.

Microscopical examination. No casts.

A few leukocytes and small epithelial cells (perhaps 7-8) in two microscopical fields. The child seems in very good physical condition, although a little pale—but no more so than her brothers and sisters.

She goes to school regularly, plays with the other children, and her parents consider her perfectly well.

History after operation. No more edema, headaches or uremia after discharge from hospital. The patient for a year past has been in perfect health and has taken up her school life.

Result. A cure. Patient's general health all that could be desired, and urine practically normal.

Regarding the indications for operative treatment in acute and chronic nephritis, I quote from my former article as follows: "During my professional career extending now over 30 years, I have made the following observations regarding kidney infection: Nephritis following acute infectious diease in children has a tendency to complete recovery. Children who survive the acute stage but continue to show albumin and renal elements for several months also frequently make a complete recovery. In a certain percentage of cases recovery does not take place. I have records of 7 of my patients who developed nephritis following scarlatina—diphtheria—measles, under my personal observation and who subsequently died of kidney insufficiency. One at the age of 5; one at 6; two at 10; one at 15, and two at the time of the first confinement after marriage at 21 and 22 years of age. In view of the uselessness of medication in chronic nephritis the proposition to treat Bright's disease surgically should be met without prejudice.

From my observations in this case and in other cases which have come under my notice I would not hesitate to advise inspec-

tion of the kidneys through lumbar incision in cases in which an acute nephritis not secondary to heart lesions does not clear up in a reasonable time—say 6-8 months—and would furthermore advise splitting of the capsule or decapsulation of one or both kidneys should they appear swollen and enlarged with the hope of preventing an acute nephritis from becoming chronic and incurable. The same procedure is, of course, indicated in acute nephritis with complete suppression of urine. Edebohls claims that his operation is beneficial by reason of an increased blood supply. This is not as yet proven. I believe that some of the virtues of decapsulation of the kidney are due to massage incident to handling of the infected organ.

753 Madison Avenue.

DISCUSSION.

DR. KERLEY.—I should like to ask Dr. Caillé what effect this operation of renal decapsulation would have on the susceptibility of the kidney during an attack of scarlet fever, or other infectious disease.

DR. CAILLÉ.—I cannot answer Dr. Kerley's question. The whole matter of renal decapsulation is so new, and we possess so very little definite knowledge on the subject of the increased susceptibility of the kidney in infectious diseases, such as scarlet fever, that I am unable to give any positive answer. There are quite a large number of children that have been operated upon, but the reports have not as yet been published because the physicians are waiting for results. In the case reported I am convinced that the child has been much benefited by operation, but whether this was due to increased blood supply or to the handling of the organ I do not know. In fact, the small hemorrhages into the tissues may have had some effect in improving the circulation.

DR. PUTNAM.—I would ask Dr. Caillé if this is the only case he has followed.

DR. CAILLÉ.—It is the only case I have studied.

DR. PUTNAM.—Were all the other cases entirely relieved of their symptoms?

DR. CAILLÉ.—When I last saw Dr. Edebohls he was then preparing a paper on renal decapsulation, and out of 80 cases operated upon he reported 10 or 12 absolute ideal cures and 8 or 10 probable cures. These latter were the cases that gave a clear urine almost all of the time with only an occasional trace of albumin.

DR. LA FÉTRA.—The improvement in these cases of renal

decapsulation is very difficult to explain. It is certainly not due to an increased blood supply to the cortex of the kidney. Experimental tests of the operation have been made by Hall and Herxheimer in England, and by Van Cott in this country. When at later periods the kidneys have been removed and studied, there has been found a new capsule with no arterial anastomosis, and a tendency for the new capsule to become thicker, and for fibrous bands to project from the capsule into the cortex, thus producing an interstitial nephritis.

Similarly in old cases of renal decapsulation for nephritis a new thick fibrous capsule is formed with no increase of blood supply to the kidney; moreover, there is no regeneration of the damaged cells of the tubules nor of the glomeruli. Relief of tension may explain; but no matter what the explanation the important point is to follow these cases to learn how they behave clinically. Probably a more careful selection of cases for operation can be made, so that decapsulation is practised only in those cases that are of local or bacterial origin, and in those in which there is an increase of tension, as in large white kidney.

DR. CAILLÉ.—Only a few weeks ago I saw a redecapsulation performed on a patient; the first operation had been done two years ago. Both kidneys were operated on for chronic nephritis. Acute uremic symptoms developed which called for the second decapsulation. I saw both kidneys and there was no fibrous change, or fibrous tissue formation at all. After the first decapsulation the kidney was about the same consistency.

DR. FORCHHEIMER.—In a certain number of cases of exophthalmic goitre, splitting the capsule of the thyroid gland brings about normal functional activity of the gland without producing any anatomical changes. A number of cases have been reported in which the benefit was not merely temporary, but the condition was absolutely cured. Whether this was due to a change of tension or to absorption of certain antibodies after the capsule was split, I am not prepared to say. To me this is the only tenable explanation to account for increased functional activity of the kidney following renal decapsulation.

DR. CAILLÉ.—In answer to Dr. Forchheimer's reference to splitting the capsule in exophthalmic goitre, I do not believe that anyone could say that splitting the capsule relieved the tension, although it might be that the bleeding alone might be sufficient to influence it favorably. So far as experience goes, the splitting of the capsule and the handling of the organ which accompanies this operation produces the good results. I believe that the massage of the organ in decapsulation is a very important factor in the relief that follows.

Surgical treatment is only indicated when there is kidney insufficiency. My interest in this was purely a clinical one, and have seen better results from decapsulation than from medication.

ACUTE MYOCARDIAL INSUFFICIENCY IN SOME INFECTIONS IN CHILDREN.

BY F. FORCHHEIMER, M.D.,

Cincinnati, O.

The term, chronic myocardial insufficiency, was introduced by Ebstein, as applying to all those conditions in which the normal relation between pulmonic and systemic circulation is disturbed on account of some anatomical change in the myocardium. In acute myocardial insufficiency the same definition holds good, except in that the process is an acute one. The importance of our subject lies in the fact that a large number of these acute myocardial insufficiencies develop in connection with the infections in childhood; scarlatina, diphtheria, rheumatic fever, septicemia, typhoid fever and pneumonia. Beyond the possibility of a fatal termination from the acute myocardial condition, there remains another which is very far-reaching for the future of the individual who survives. In the greatest number of cases of heart weakness developing during pubescence and young adult life, the only explanation that has presented itself is heart strain, either due to development or inherent, *i.e.*, congenital, myocardial weakness. In some of the diseases mentioned before, especially diphtheria, scarlatina, typhoid fever, the myocardial changes, especially the interstitial ones, are so well marked that permanent danger is done to the heart which may only manifest itself when an extraordinary call upon the functional activity of the heart is made. The view which is daily gaining more ground, that socalled idiopathic dilatations of the heart are not so common as was supposed, therefore, is probably true; the basis for a dilatation being some myocardial change. As it is a fact accepted on all hands, that, in every case of peri- or endocarditis, the myocardium is more or less involved, it will be seen how great the number of cases becomes in which it is possible that acute myocardial insufficiency may develop. In pericarditis with effusion the additional element may be the possble failure in diastole; in endocarditis, the chance that

a damaged myocardium may not be able to perform compensation for a valvular lesion. Furthermore, in acute nephritis of scarlatina, the increased resistance in the left heart will be less readily overcome by a damaged than a healthy myocardium. But the development of symptoms of acute myocardial insufficiency depends upon the amount of damage done to the myocardium, so that in a number of cases no symptoms develop at all; in a much larger number the symptoms develop after the febrile course of the primary disease has ceased, being due to a continuance of the process and its bad results; in these cases the development of symptoms may be postponed indefinitely; in a certain number of cases the changes in the myocardium are so great that they manifest themselves early during the febrile period.

Of the frequency of myocardial changes in connection with infectious diseases there is no doubt since the adoption of Krehl's method for examining the heart. E. Romberg has found myocarditis present in 6 out of 11 cases in typhoid fever (ten children); in 7 out of 8 cases in scarlatina (six children); 2 cases of scarlatinal nephritis and in 4 cases out of eight in diphtheria (children); in 4 cases examined by Ribbert, myocardial changes were found; in the latter disease myocardial changes are referred to by a host of authors, French, German, English and American, beginning with Bristowe, in 1859, and ending with Heubner, in 1903. Arthur Neumann (1900) examined seventy children for dilatative heart weakness, amongst which number he found eight affected. I was interested in reading the short histories appended to the article to find that six had had infectious diseases; these had had diphtheria alone or diphtheria and other diseases, one scarlatina alone; one had tuberculosis, which need not be considered, and in one only there was a negative history of some infection.

The mechanism of the production of acute myocardial insufficiency is very simple, then; some damage done the myocardium; immaterial whether primarily an interstitial myocarditis (E. Romberg) or primarily a degenerative process and secondarily an interstitial process (Ribbert). Nor does it interest us for the present to discuss whether it be the toxins or the bacteria which produce the troubles (Lees). When a sufficient change takes place in the myocardium so that it becomes insufficient, then the reaction on the part of the heart as a whole is sufficiently well marked to assume a distinct clinical picture. At first there is

noticed a diminution in heart force and arryhthmia; then follows dilatation of the left heart, then of the right heart, with relative insufficiencies of their valves; as soon as the right heart is seriously affected, marked enlargement of the liver. I have seen the latter descend to the level of the umbilicus. Sometimes the right heart is affected first; but not commonly, as affections of the myocardium follow the rule of all acquired, noncongenital diseases of the heart, viz., that they are more common on the left side. If to a damaged myocardium there be added a predisposing cause, especially affecting the right heart, as whooping-cough or pseudopertussis, the right heart first becomes insufficient. The signs and symptoms vary in different diseases, as will be shown further on, but upon the whole there is produced weakness, irregularity of pulse and heart impulse—tachycardia or bradycardia; a broadening of the heart dullness and bruits heard most commonly over the apex, the mitral and the tricuspid area, but frequently, also, over the base. To this class belong the 2 cases I referred to in my contribution to Jacobi's Festschrift; in 1 case the myocardial change was obesity; in the other it was due to three attacks of influenza, for which disease Huchard has established myocardial changes.

Diphtheria, of all the acute infections, most frequently produces myocardial insufficiency. Schmaltz claims that acute myocarditis occurs in from 10 to 20 per cent. of all cases of diphtheria; fortunately, not all of these lead to myocardial insufficiency. The process of myocardial insufficiency, which has been confounded with other methods of heart death in diphtheria, does not begin before the end of the first week of the disease, is most common during the second and third week and may develop as late as the sixth or eighth week. The symptoms are those already described, *i.e.*, when there is only diphtheritic myocarditis. The patient dies when the diseased myocardium can no longer overcome the demands made upon it, especially from distension; primarily then from heart paralysis; but in addition, secondary effects upon innervation and possibly, upon oxygenation, although the latter is rare, as is evinced by the absence of cyanosis. But not enough stress, in my opinion, can be laid upon the fact that pure myocarditis is not the only cause that produces heart death in diphtheria; there are at least two other processes that complicate it. The first of these is the effect of the diphtheria toxin upon the cerebral vasomotor centre first discovered by a series of in-

geniously conducted experiments by Romberg and Pässler and fully verified by Heinike, Rolly, v. Stejskal. As the result of this action, paralysis of the vasomotors takes place; therefore, dilatation of the blood vessels, with the result that the blood is not propelled normally. Unfortunately, the parts supplied by the splanchnic nerves will contain so much blood, the liver alone containing one-quarter of the whole amount of the blood in the body, that circulation becomes an impossibility. The result is, that the heart simply "bleeds itself" into the splanchnic area, and when, after a time, very little or no blood flows into the right heart, all of it being stored up in the vessels of the abdominal cavity, systolic contraction ceases. Again, here the symptoms are fairly characteristic. In diphtheria it is the form of heart death that occurs very early in the disease, as I have seen it as early as the third day; this before the days of antitoxin. The dominant symptom is increasing rapidity of the pulse, not marked changes in rhythm, but continuous diminution in filling of the arteries, so that the pulse becomes weaker and weaker, the arteries more and more empty, until it no longer can be felt. The condition of the heart is negative; no dilatation, no bruits, no stases anywhere, therefore no enlargement of the liver. But, again, here a continued diminution in force and a constant increase in rate; this is characterized by dullness of heart sounds, especially the first; and, finally, by a cessation of the heart's action. The aspect of the patient is more or less characteristic; pale, more or less restless, no brain symptoms, *i.e.,* no unconsciousness. The patient feels weak and is unwilling to move. This form of heart death, in pneumonia, I have seen in infants; the abdomen becomes much enlarged. Romberg and Pässler have shown that the diplococcus pneumoniæ produce the same results upon the vasomotors as the diphtheria bacillus.

The whole process, when thoroughly developed, runs its course in a short time, from 6 to 18 or 24 hours.

The second method, complicating the clinical picture, is an affection of the vagus. At the present day the myocardial origin of symptoms, following the physiological research work of Gaskell and of Engelmann, is so much in the ascendant, that the origin of symptoms, because of affections of the vagus, is rather frowned upon. The most recent physiological investigation of Pawlow, in that he kept dogs alive for over five months after bilateral vagotomy without producing changes in the heart, seems to con-

firm this view. But that the vagus does have an effect upon the human heart is shown by the experiment of Czermack upon himself. But even Engelmann shows that nervous influences play a decided rôle in influencing the number of cardiac contractions, and Lommel, in a most exhaustive paper on arrhythmia, has come to the conclusion that "organic heart diseases never produce it"; they are simply neuroses and, as he has seen it, may be produced by irritation of the vagus centre in diseases of the brain. The latter point is especially valuable, as he says, in permitting us to differentiate organic diseases of the heart from functional ones. It is thoroughly established that, in diphtheria, the vagus is frequently affected, and it is also thoroughly accepted that in a large number of infectious diseases we have arrhythmia. If these two facts be put together with the new ones established by Lommel, we can certainly not be far wrong in stating that in diphtheria arrhythmia may be due to disturbance of the vagus tonus. The next question that arises is the possibility of producing heart death by vagus affection in the human being. It must be confessed that we have no evidences upon this subject, for Czermack, after having caused his heart to stand still by pressure upon his vagus, always came to again. But the little that has just been said will certainly justify the conclusion that, even if the vagus may not produce heart death, the effects of its irritation in a myocardially insufficient heart may hasten the fatal outcome. For therapeutic purposes the fact that myocardial insufficiency in diphtheria may not be a pure process, but complicated, at least, by the two other factors just discussed, seems to me to be very important.

Next to diphtheria my own experience with acute myocardial insufficiency has been most extensive with the septicemia of influenza. Here again we have a complex process, the result of infection with the influenza bacillus and secondary infections with pus producers, notably streptococcus and the various staphylococci; in by far the greatest number of cases the secondary infection coming from the respiratory tract, but sometimes from the gastrointestinal tract. I have seen no case in connection with influenza in which the mechanism of Romberg and Pässler seemed to play a very important rôle, although the influenza toxin seems to have a direct effect upon the vasomotor, as is shown by the change in the pulse temperature ratio. All the cases I have seen have been accompanied by more or less cardiac change. The

worst case in children that has come under my observation which has recovered I herewith report.

A. A., seen in private practice, *aet.* seven years. Family history, tuberculosis on mother's side, neuroses on father's side. The child was bottle-fed as an infant with normal results; she has had one previous attack of mild influenza; otherwise she has been well. On October 27, 1902, I was first called to see her. There was present a house epidemic of influenza, she being the last one of the children to come down with it. It was the ordinary respiratory form, but in a short time developed into pseudopertussis, for which she received quinia hydrobromatum with good results as far as the number of attacks was concerned. On the morning of November 7th I was again called to see her. The statement was made that she had passed a restless night and had seemed very feverish. Upon examination there was found a temperature of 105.4°F., pulse 122 and respiration 34. No evidences of any organic lesion, even the cough had disappeared. Notwithstanding hydrotherapy, the temperature reached 106.5°F. that evening, with a pulse rate of 140 and respiration 40. The patient became unconscious, took medicine with difficulty, became jerky and was very nervous. At eleven in the evening she became partially conscious, the temperature having been reduced to 104°F., pulse 120, respiration 32.

November 8th. May be summed up as beginning with a temperature 102.6°F. in the morning, the pulse remaining at 140, and the respiration 32. The maximum of temperature that day was 105.4°F., pulse and temperature not varying much from the morning's rate. The nervous condition improved, as the temperature came down the cough began to reappear, all other functions being normal. The urine contained a small quantity of albumin, nothing being found microscopically. Throughout the course of the disease there was no evidence of nephritis. On my evening visit I found the heart dullness somewhat increased, no bruits. At noon massive doses of unguentum Credé were used. The night was better than the former one, but still some delirium upon awakening.

November 9th. Found the patient with a morning temperature of 99.2°F., pulse 98-105, respiration 24. The maximum temperature was 100.2°F., pulse 100-104, respiration 24-26. Cough has reappeared, nervous symptoms gone, but the pulse was decidedly irregular. For this reason stimulants were given. Increase in heart dullness seemed more marked than before.

November 10th. The temperature in the morning was subnormal, pulse from 84-88 and intermittent, respiration 24. From this time on for nearly two months we had subnormal temperature in the morning. The pulse even on this day sank to 72 and rose to as high as 96. The heart as before.

November 12th. I found the child pale, cyanosed, breathing with difficulty and sweating profusely. Pulse was irregular, from 74-78, and heart exertion would bring it up to 100, the pulse becoming emptier and more irregular. Examination of the heart revealed heart dullness beyond the right border of the sternum, left auricle enlarged, systolic bruit at the apex, and a diastolic bruit at the base. Liver enlarged so that its free border could be felt 2 cm. below the free border of the ribs and painful upon pressure. By means of absolute quiet, stimulants, codein for the cough, strophanthus, then resistant movements, the child was able to sit up in bed on November 21st for the first time. During this time the pulse varied from 64 to 88, the former representing a bradycardia in a child. The peculiarity of the pulse, as far as rate is concerned, is best represented by the record on November 21st, when she was allowed to sit up in bed. Under observation for an hour, the pulse rate, counted every fifteen minutes, was 72, 76, 78, 78. When she sat up in bed the second time, on the same day, the pulse varied from 88 to 100, showing the peculiar irritability of the heart's muscle. The hepatic sign of myocardial insufficiency lasted only eighteen hours. So unreliable was the heart's action, although the dullness had diminished and the bruit at the base had disappeared, that I did not venture to allow the child to get up until December 6, 1902, about one month after the onset of symptoms; permitting her then to sit up in bed before allowing her to get up, for three hours at a time, her pulse being watched at regular intervals. In the beginning of her period of sitting up in bed, if, as was natural in a child of her age, notwithstanding all precautions that had been taken, she would move rapidly, her face become pale, she would again sweat freely and the heart dullness would again increase. On the day before she got up, being allowed to sit up in bed for three hours, the pulse was 98 at the beginning and 80 at the end of the time. It is interesting to note here, that for some time any exertion would manifest itself by a reduction in pulse rate; this lasted for eight days, and it was only after this manifestation had disappeared, that I felt safe in pushing the heart to

further exertion. On December 23d she was allowed to walk out of her room for the first time, and by this time the bruit at the apex was very faint, no enlargement of the heart could be detected. On Christmas day she was allowed to walk downstairs, her pulse now varying from 90 to 94, but still intermittent; in five days after this she was allowed to take a drive; January 9, 1904, she was allowed to walk upstairs and was then discharged. I took occasion to examine her occasionally and found her pulse still continuing to intermit, the bruit gradually disappearing and the heart losing its irritability. I examined her for the last time on May 17, 1904. She goes to school, romps about with the other children, does not have dyspnea on exertion and seems to be perfectly well. Her heart is of the normal size, there is no bruit anywhere and none can be brought out by exertion. The mother tells me that her pulse still intermits occasionally, but I could not find it so after having examined it for five minutes.

According to my view, we here had a myocarditis, producing acute myocardial insufficiency as the result of a septic infection engrafted upon an influenza. The bruits were due to relative or muscular insufficiencies, the great irritability of the heart especially speaking for myocardial trouble. It seems to me that this can be looked upon as a pure case of myocarditis, although the possibility of an endocarditis cannot be absolutely excluded. Contrary to Romberg's view, I believe that all the symptoms can be explained upon the basis of this assumption; even the bradycardia which we know to be due, at times to fluctuations of intracardiac pressure, diminution (Ludwig and Thiry) or great increase (Heidenhain) may be explained on this assumption. The absence of edema, one of the points that Romberg insists upon as militating against the existence of myocarditis in influenza, is explained by the fact that the myocardial insufficiency is not persistent. The pallor and sweating is due, not to primary vasomotor disturbances, but to sudden changes in the centres, the result of the dilatation of the heart. But the fact that other processes interfering with circulation exist in influenza cannot be denied.

It would extend this paper beyond the proper limits to discuss acute myocardial insufficiency in connection with scarlatina, typhoid fever or rheumatic fever; in all these affections myocarditis is not uncommon, acute myocardial insufficiency, however,

is rare in children; although I have seen one fatal case in scarlatina with scarlatinal nephritis.

Our treatment has been added to since Romberg and Pässler's investigations have been accepted, but only when this complication exists. For uncomplicated myocardial insufficiency the first principle is absolute rest. The digitalis group of drugs may be used; their efficacy depends largely upon the amount of myocardium affected. Predisposing causes should be controlled; in the case reported it was the pseudopertussis which was easily controlled by very small doses of codein. Stimulants will be required in most of the cases from alcohol to camphor or ether, according to the severity of the case. The convalescence must be especially guarded and, for the purpose of strengthening the myocardium, mechanical means are invaluable: Swedish movements, massage, vibratory methods. The diet is the same as in all myocardial affections. Laxatives that act violently should be avoided. Strychnia may be used for its general tonic effects; except in toxic doses it has no effect upon the heart nor upon the vasomotors. Nitroglycerin should never be used, unless there be anginous attacks; at all events, care must be taken to exclude the existence of vasomotor paralysis, when it does harm.

For the vasomotor paralysis Pässler has shown experimentally that digitalis produces only very transitory results, no effects upon the blood vessels; no effect is produced by alcohol or ether, possibly a bad one on the vasomotor centres; ergotin is without value; camphor produces good results, but the best are produced by caffein. Saline infusion produces temporary results and abdominal massage fairly good ones.

My own method, which I have used with good results in 2 cases, is as follows: hypodermic injection of adrenalin; this drug has an elective effect upon the blood vessels supplied by the splanchnic nerve. Its effects are very transitory; therefore, the injections have to be repeated every two hours. Saline transfusions are used in the beginning in order to keep up the heart's contractions. Ice-bags are put upon the abdomen; stimulating the splanchnic reflex. In one child I was able to bring the pulse down from 140 to 60 by the use of these alone. After the most violent symptoms have passed over, then caffein soda-salicylate is given, every two to four hours, alternating with the adrenalin, which is gradually dropped. The convalescence is treated as is indicated in the case reported.

DISCUSSION.

DR. ROTCH.—I hope that the real nature of these cases of acute dilatation seen in young children will soon be cleared up. All have seen such cases which have lasted but a short time. I have treated these cases with absolute rest as advocated. I believe that, as a rule, the general practitioner does not recognize these cases.

DR. JACOBI.—It has only been within the last two dozen years that the myocardium has received much attention. Our forefathers simply made a diagnosis of heart inflammation and let it go at that. With better means of diagnosis we went a little further and diagnosed pericarditis and endocarditis, but myocarditis was seldom thought of. It has been only within the last ten or twelve years that the profession has awakened to the necessity of studying the myocardium and its diseases more profoundly.

As a matter of fact, in dealing with any severe case, we are not dealing with an uncomplicated condition. It is not infrequent now to find endocarditis, pericarditis and myocarditis existing at one and the same time; autopsies show this to be true. It has been particularly so since Englemann and others showed the importance of the heart muscle when removed from the influence of the nervous system that more attention has been paid to the changes in the muscle itself.

There are two forms of myocarditis recognized, viz.: (1) simple parenchymatous degeneration, and (2) the real interstitial inflammation. Now and then one sees an old cicatricial condition more or less extensive. Parenchymatous degeneration shows itself in different ways. A few years ago we were told by Hektoen, of Chicago, that this degeneration went as far as fragmentation; that Dr. Hektoen himself recognized as a mistake, occasioned by his mistaking the postmortem for antemortem changes. This fragmentation should no longer appear in the text books. There is, however, a parenchymatous change resulting from the action of bacilli or toxins in infectious diseases which is spoken of as inflammation, as a myocarditis. We see such changes after every infectious disease. Dr. Forchheimer has questioned the possibility of attributing it to bacterial action or to toxins; there is one fact we are all acquainted with—that there are more toxins than bacteria that gave rise to the symptoms. In pneumonia we know the particular danger is not so much during the acute stage in many cases as when the crisis comes; then at once the temperature falls, the pulse falls and becomes irregular, and the picture changes; if the patient dies on the fifth, sixth or seventh day it must be due mostly to the toxins that have developed during the course of the disease with their particular influence on the heart.

In reference to the enlargement of the liver Dr. Forchheimer has correctly stated that, if the right heart were chiefly involved, the liver would swell. The liver would be found swollen to a

certain extent in cases of many or most acute diseases of childhood. When we have heart symptoms, with a swollen liver coming on rapidly, with slight edema about the hands and feet and also about the face for the first few days, one can be pretty certain that there is a myocarditis existing. The large swollen liver is associated, when at the beginning of the disease, with a myocarditis and not so much with an endocarditis. In myocarditis it is characteristic to find a moderately sized heart in cases where the cardiac symptoms have existed for years. Sometimes in myocarditis the heart symptoms are localized more about the mitral than about the aortic valve and frequently with a murmur, this is why incorrect diagnoses have been sometimes made. If the murmur has existed for a long while, no matter whether it was localized or universal, if there was no increase in the size of the heart, it means a myocarditis. There are more cases of myocarditis with no enlargement of the heart than with it. On the other hand, endocarditis with valvular affection will soon lead to dilatation and hypertrophy.

The questions regarding the toxins or bacteria will possibly be changed in a few years. Vaughan and Ehrlich believe that bacteria are not single bodies. Vaughan recently showed me two bottles of different toxins made from the same bacillus. Ehrlich believes the poison of diphtheria is not one but two, and he attributes the paralysis of diphtheria to a poison quite different from that which gives rise to the first symptoms. We are no doubt a long distance from the end of our knowledge regarding this.

I do not agree with Dr. Forchheimer in regard to treatment. If there is anything that I dislike to give in the beginning of acute myocarditis it is digitalis. Digitalis puts too much labor upon the heart; and so does strychnin. In chronic myocarditis I have found that strychnin makes the patients worse and opium is better. In acute myocarditis I rely upon cold applications to the heart, the ice-bag for a short time, not for a long time in young children. I also place much confidence in codein and in the iodids. I do not dislike the nitrites when the arteries are poorly filled. Absolute rest is enforced. In myocarditis with danger from overstrain I should rather rely upon some opiate, iodid or nitrate. There is no better thing than rest in bed for weeks and months. Place the patients in bed in a cool room, open the windows and give them absolute rest.

DR. FORCHHEIMER.—In closing the discussion, I would say that the object of this paper was to establish some form of treatment in myocardial insufficiency, which could be applied in the acute as well as the chronic cases. I have tried to establish two clinical pictures, first myocardial insufficiency, due to an acute myocarditis as a pure process and, second, myocardial insufficiency as a complication.

Dr. Jacobi and I differ only on the surface. I have not taken

up the treatment of myocarditis. I agree with what he has said regarding the treatment of myocarditis. I rarely use digitalis there and also place the utmost reliance upon rest. Digitalis should never be used except for the specific indication of myocardial insufficiency, acute or chronic, and its value in this condition is dependent largely upon the quantity of healthy myocardium it can affect.

DR. JACOBI.—I feel sure that Dr. Forchheimer would not speak of insufficiency of the heart as a functional trouble. We have used this phrase "functional trouble" too often and I think I shall not use it so frequently. I regard the particular cause of the disturbance as situated in the structure of the muscle.

THE ACUTE PYELITIS OF INFANCY, WITH REPORT OF A CASE.

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Any disease of infancy which can pass unrecognized, while under intelligent observation for several weeks, on account of the absence of local symptoms pointing to its location, deserves to be called frequently to the attention of the profession in order that the characteristics of its manifestations may be familiar, and that subsequent cases may not be overlooked. This is particularly true of any disease which will continue indefinitely without specific treatment, but may be readliy cured by the use of appropriate remedial agents.

The acute pyelitis of infancy is a disease of this sort. Its rarity is sufficiently emphasized by the fact that Holt has reported only 3 cases; Heubner, 2 cases; and Thompson, of Edinburgh, 8 cases.

The etiology of most of these cases is very indefinite. The fact that a large proportion of the cases have occurred in female children makes it seem probable that the infection is often through the urethra while the organism associated with most of the cases, the bacillus coli communis points to an infection originating from the alimentary tract, as does the fact that many of the cases follow intestinal disorders.

The clinical manifestations give no indication that the pelvis of the kidney is the source of the trouble. They usually have abnormal movements from the bowels, associated with a high temperature of irregular type with marked remissions and followed by rapid elevation, which in some cases is accompanied by chills. There is thus a great likelihood that these cases may be treated as cases of intestinal disorder associated with a high temperature, or that they may be considered of malarial origin.

The procuring of a specimen of urine from an infant involves some difficulty for those unaccustomed to work with these little ones, so that this one method of making a diagnosis in this condi-

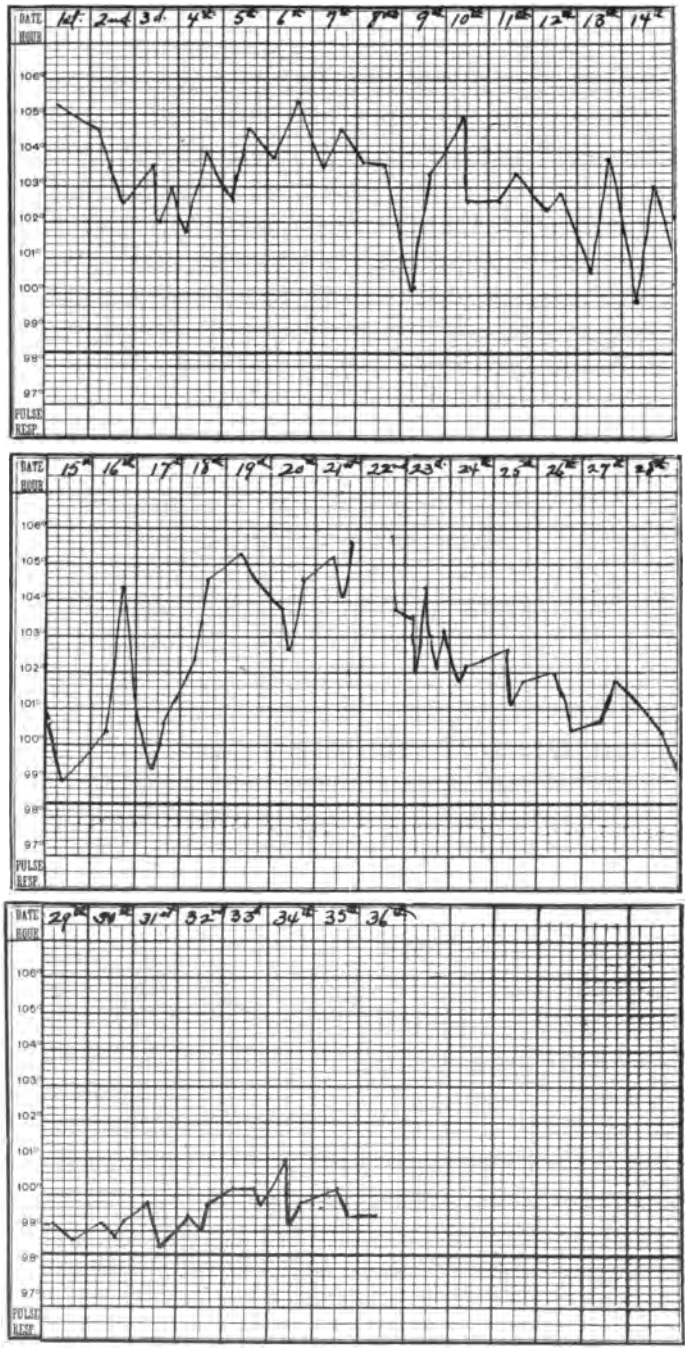
tion is very apt to be neglected, and, again, pain referred to the region of the kidneys is not a symptom that can be relied upon in infancy. On this account it has happened that many of the recorded cases have been allowed to progress for long periods before curative treatment has been used. This is the more unfortunate since the curative treatment is very simple and safe—the administration of alkalies in order to neutralize the reaction of the urine from the extreme condition of acidity always found in this disease—such treatment, so far as I know, having always affected a moderately rapid cure.

The following case seems of interest since it occurred in a male infant which, on questioning the mother, was found to have shown some evidence of urethral irritation at the beginning of the attack, which attack was preceded by two or three weeks of slight digestive disturbance as indicated by abnormal stools. This child continued sick for three weeks, with an irregular temperature which, toward the end of this period, remained constantly very high and was associated with nervous symptoms, such as twitchings and stiffness of the back of the neck. As soon as remedial treatment was instituted, on the twenty-second day of the disease, the temperature steadily declined until it reached normal on the twenty-ninth day. The child has since steadily gained in weight and become perfectly healthy.

A detailed history of the case, with the results of the examination of the blood, follows:

History.—Male child, eight months old, fairly nourished, suffered from slight digestive disturbance for three weeks, after which it was suddenly taken very sick on the eighteenth of February and had a high fever, 105.2° F., and its movements became green. Its food was stopped, it was given calomel and all the water it wanted. During the following twenty days the child was fed for the most part on barley-water and egg-albumen water. The administration of milk was usually followed by a higher range of temperature. Medication was directed for the most part to the correction of gastroenteric symptoms and the reduction of temperature; the medicines used were calomel, castor oil, bismuth, aconite and acetanilid and brandy. Irrigation of the bowels was used occasionally. The variation of the temperature may be seen on the chart.

Twenty-second day.—On this day the baby was brought to me for treatment. It now has some color in its cheeks, which are



TEMPERATURE CHART OF A CASE OF ACUTE PYELITIS OF INFANCY.

thin, and it has a large, broad forehead. Head circumference, 42½ cm. Chest, 41½ cm. It has had some twitchings of the face and has cried out in its sleep. There seems to be a little stiffness of the back of the neck. Ears negative. Respirations 80, but there is no swinging of the alæ nasi. No expiratory grunt or pause between inspiration and expiration. The lips are somewhat cracked, the tongue is very slightly coated. The cervical lymph nodes are not enlarged. The heart and lungs are negative. The liver is not enlarged, spleen is not enlarged, abdomen is not distended. No diagnosis was made. The urine and blood had not been examined. Ordered 3 drams of castor oil, 1 grain quinin, bisulphate in solution every two hours. Milk stopped, a proprietary food ordered. Evening temperature, 105.7°F. A bath at 95°F. ordered, temperature reduced to 103.8°F.

Twenty-third day.—Morning temperature 102°F. Urine acid, specific gravity 10.10, contained a large amount of pus, very few hyaline casts with trace of albumin.

Blood examination by Dr. F. C. Wood: 3,900,000 red corpuscles, 70 per cent. hemoglobin, 13,000 white cells, 67 per cent. polynuclear neutrophiles and 33 per cent. lymphocytes, large 2 per cent., small 31 per cent. No eosinophiles or mast cells seen in 200 leukocytes. No nucleated red cells found. The baby had been sleeping for four hours, so had no digestive leukocytosis.

During this day the baby had a considerable stupor and in the morning a rise of temperature to 104.4°F. Ordered urotropin ½ grain every four hours. Ordered irrigation of bowels every morning and poultices twice a day over kidneys.

Twenty-fourth day.—Baby had a fair night, has taken 11 ounces of nourishment. Ordered solution of citrate of potash continued, 2½ grains every two hours to eight doses in twenty-four hours. Temperature to-day not above 102.5°F. Urine now alkaline, but amount of pus four times as great as yesterday.

Twenty-fifth day.—Temperature lower and amount of pus in urine much less than two days ago. Urine planted in agar and pure culture of bacillus coli communis obtained.

Twenty-sixth day.—Temperature ranges between 99.4° to 102°F. Child brighter, but hands are cold and heart intermits every third beat. Ordered urotropin stopped. Child put on modified peptonized and pasteurized milk containing 3 per cent. fat, 6 per cent. sugar and 2 per cent. proteid.

Twenty-seventh day.—Temperature range much the same as

yesterday. Child seems brighter. Pulse regular. Respirations still 60 to 80. Pulse 120. Amount of pus in urine markedly less. Ordered one dose of ½ grain of urotropin at night.

Twenty-eighth day.—Heart skips every third beat, hands cold and bluish. Abdomen distended. Movements contain large curds. Stopped urotropin, ordered 2 drams castor oil. Stopped modified milk, ordered proprietary food. Toward evening child seems more comfortable. Temperature 100.4°F. Twelve ounces of nourishment taken.

Twenty-ninth day.—The castor-oil was followed by a large movement containing much mucus and large curds and after this the child rested much better. Respirations 62 to 80. Pulse 120. Temperature about 99°F. Urine clearer. Ordered citrate of potash 5 grains eight times a day. Sixteen and one-quarter ounces of nourishment taken. From this time the child was kept on the citrate of potash and fed with modified milk, of which it took daily an increasing amount until on the thirty-seventh day it took 36 ounces. Some leukocytes persisted in the urine. The respirations continued about 70 a minute. The parents were then advised to take it to their home, the same treatment being kept up. It has continued to improve without any remissions.

DISCUSSION.

DR. MORSE.—It seems to me that the frequency with which we meet this disease depends upon the frequency with which we examine the urine. For several years I have made it a routine practice to examine the urine of every baby not only at the hospital, but also in private practice. During this period I have had from 2 to 4 cases of acute pyelitis in every service. Even with an examination of the urine I believe that the diagnosis is often rather difficult. Very often an acid urine is found containing a great deal of pus and very little else. In such cases it is very hard to tell whether the pus came from the kidney or from the bladder. In the cases in which there are squamous cells I suppose the trouble is in the bladder; when there are spindle or caudate cells I believe that the trouble is either a pyelitis or a pyelonephritis. The presence of casts of course confirms the diagnosis of pyelonephritis. It is my impression that most of these cases are due to the colon bacillus.

During the past few months I have seen 2 cases which have opened a new field to me. In 1 case pus was found in the urine and a cystitis rather than a pyelitis was suspected. Dr. Rotch, who

saw the case in consultation, suggested the possibility of tuberculosis. Further examination disclosed bacilli in the urine, which answered all tests for the tubercle bacillus and killed guinea-pigs. Later developments showed that the trouble was undoubtedly located in the kidneys. In the second case, also, tubercle bacilli were found associated with pus without other cellular elements or casts. I have found little difficulty in obtaining specimens of urine from babies, especially in hospital work. For the boys a small, wide-mouthed bottle is used and found very satisfactory. Various methods are used for obtaining the urine from little girls, among these, short confinement in the Bradford frame, a soft ring lined with dental rubber to make a basin, and a peculiarly shaped tin receptacle.

DR. ABT.—Several years ago Escherich and his students studied colon bacillus infections of the bladder and they reported quite a number of cases. Some of these infections followed intestinal disease, more particularly follicular enteritis.

The colon bacillus probably enters the urinary organs from without, or through the vascular or lymphatic systems. Since the report of Escherich's cases appeared, the number of reported cases of pyelitis and cystitis is increasing in the literature. During the past winter I saw a case very similar to Dr. Freeman's, which was of great interest. The patient, who was less than a year old, and had been previously well, was taken severely ill three weeks before I saw him, with a high temperature frequently reaching 105° or 106° F. The temperature was irregular in character. Some days it was intermittent and other days remittent. The little one was desperately ill; the emaciation was extreme and prostration was marked. The examination of abdominal and thoracic organs was negative, though the urine, which was acid in reaction, contained large quantities of pus, a few hyaline and occasional granular casts. The interesting feature about the urine, which was brought out in the bacteriological examination by Drs. Le Count and Weaver, was the finding of a bacterium which resembled in many of its morphological characters, the Shiga bacillus.

The illness ran a protracted course. The infant continued feeble for a long time, but eventually recovered. Treatment consisted in the administration of urotropin and salol, in 1-grain doses.

Another case was that of a little three-months-old baby who was sent into the hospital because it had very high fever and purulent urine. The urine contained hyalin casts and colon bacilli; it was acid in reaction and contained pus. The right kidney was prolapsed, though not movable, and could be readily felt through the abdominal wall. The child emaciated very rapidly, the pus did not diminish in amount and death occurred at the end of about two weeks.

Autopsy showed an anomaly of the urinary apparatus; on both sides double ureters were present. The bladder presented a diverticulum and the left kidney was the seat of extensive pyonephrosis with pyelitis and some involvement of the ureter.

DR. ROTCH.—Infection by the tubercle bacillus is an interesting condition. The fact of its being so often discovered with so much difficulty gives evidence of the latent character of the affection. In cases I have seen in hospital and private practice the disease seemed to be extremely latent and even when the presence of tubercle bacilli was not proved the clinical symptoms often extended over a period of weeks.

DR. DORNING.—I think the Society, in discussing this subject, cannot lay too strong emphasis upon the importance of examining the urine in every case of disease. Another method of obtaining the infant's urine should be mentioned, namely, by means of a clean, soft rubber ice-bag adjusted so that its aperture includes the genitals, and then held in place by tapes fastened around the baby's waist and under the perineum. Many years ago Dr. Jacobi had presented to the profession a small metal catheter about size No. 3, English (No. 9, French), made in two sections to be carried in the ordinary pocket case. It became so popular at my clinic, where I was in the habit of demonstrating its use, that an instrument-maker in the neighborhood obtained the pattern and in a short time disposed of several hundred of them. In the use of a soft rubber catheter it is important to select one of good quality, particularly on account of its small caliber. I recall one case in which a poor rubber catheter broke during its withdrawal from a male urethra, leaving an inch of its tip in the bladder.

The diagnosis of pyelitis is not always easy, the clinical symptoms being at times somewhat vague. Now and then I have found pyelitis giving symptoms that led me to suspect bladder difficulty, such as frequency in urination and distress at the neck of the bladder. Examination in some of these cases showed a normal bladder, but later on the evidences of pyelitis became more distinctive. I think one should suspect a tuberculous condition in the majority of cases of pyelitis. As already mentioned the condition is often found to be latent. Very often the microscopical examination does not help us, and, not infrequently, the inoculation of guinea-pigs will give negative results. With regard to the use of urotropin there is one point in connection with Dr. Freeman's case that I would consider, and that is the use of an alkaline salt to neutralize the acidity of the urine at the same time he was giving urotropin. I think it is now generally acknowledged that urotropin acts as a urinary antiseptic only in acid urine, and, hence, to obtain this effect it was well to render the urine slightly acid by administering sodium benzoate in conjunction with urotropin when the indication exists. I would ad-

vise careful observation in cases of prolonged administration of urotropin as it sometimes has an irritating effect upon the bladder and kidneys. I have seen some patients who could not take urotropin at all. In two children I have been unable to give even ½ grain doses for more than a brief time, and in two adults 2 grain doses could not be taken without developing marked bladder distress.

DR. CRANDALL.—I should like to ask Dr. Freeman about the ultimate effects of such cases of pyelitis, and the susceptibility of the kidneys. Scarlatinal nephritis may disappear entirely and yet leave behind an increased susceptibility to kidney trouble in later years. Does a pyelitis leave such an increased susceptibility behind? I have 1 case which I have watched for nine years but, up to this time, absolutely nothing could be referred to the kidneys and the cure seems to be complete.

DR. HOLT.—I have one patient who has been well now nearly nine years following an attack of acute pyelitis, and another is well who had an attack eight years ago. The attacks were in both instances severe. The urines were examined at intervals in both cases for several years; but no relapse occurred, and no sequelæ followed.

DR. JACOBI.—A great deal is heard upon this subject and evidently very little impression is made on the general medical public; it takes a great deal of hard hammering to get things into the heads of all of us medical men. Pyelitis has been up a number of times for discussion and then is forgotten. If the urine is examined in every doubtful case more cases of pyelitis or pyelonephritis will be found. There are certain diagnostic points which are correct. If there is pus and a great deal of bladder epithelia in the urine it is reasonable to suppose that at least a part of the pus and epithelia come from the bladder. Again, if there is much renal epithelium in the urine, with hyalin casts and only now and then a small granular cast, it points to the kidney as the seat of trouble. In some cases we get blood and pus from the kidney and this indicates a pyelitis. In cases of pyelonephritis there are more kidney elements to be found than in a simple pyelitis. I have not infrequently been called to see cases of protracted fever in which the unfortunate diagnosis had been made of malaria, and brief examination revealed either a pyelitis or a nephritis. This is a subject that is always being forgotten; that pyelitis is a disease frequently found in young infants. In every doubtful case, where there is fever the urine should be carefully examined.

I am not fond of urotropin, although many children may take it well. I recall a child of three or four years old in whose urine uric acid fell down in amorphous masses. Urotropin was given a number of times and the result was that each time the uric acid no longer appeared in such masses but came down in crystals. A

remedy I like, not in the acute stage, but in the subacute and chronic stages of the disease, is gallic acid in fair doses; this is well tolerated; I remember but 1 case in which it was not tolerated. Adults can take from 100 to 180 grains a day, and a one-year-old child can take from 5 to 10 to 15 grains, even up to 20 grains daily, without disturbing the digestion. Tannic acid must not be substituted; it is not well borne.

It is difficult to discover tubercle bacilli in the urine in most cases. Those cases which are the result of bacterial infection will get well as a rule; but those resulting from stone are of a different nature.

DR. FREEMAN.—At the Foundling Hospital, in New York, the urine of young children is always examined as a routine practice, but cases of pyelitis are rarely, if ever, seen there. There is no difficulty in obtaining specimens of urine from even the youngest children. The methods mentioned by Dr. Morse may be used or, as in the case I have reported, the mother or nurse watches the child and when he or she urinates they use the cup.

My impression is that these pyelitis cases, when they recover, recover slowly and relapses do not occur.

Urotropin was used in my case before the citrate of potassium.

AN EPIDEMIC OF VULVOVAGINITIS AMONG CHILDREN.

BY A. C. COTTON, M.D.,
Chicago, Ill.

The specific character of the recent epidemic of vulvovaginitis among children in the hospital was satisfactorily demonstrated: First, clinically by a purulent, more or less copious, greenish or yellowish offensive discharge which was very intractable to treatment; second, by the undoubted infectiousness as seen in the rapid extension to other patients, in spite of ordinary means of prevention; third, by microscopic examinations and Gram's test of the discharges made by several members of the house staff in the early part of the epidemic. This was confirmed by reports from specimens submitted to Prof. Hektoen, and in the last 3 cases by the findings of Prof. LeCount, who succeeded in isolating in pure culture the gonococcus of Neisser. Mr. Baer, who did this work, gives the following summary:

"A diplococcus has been isolated in pure culture from 3 of your cases of vulvovaginitis. The diplococcus has been identified by its morphology, staining reactions and peculiar cultural reaction as the gonococcus. The medium best suited for its isolation and cultivation is a mixture of hydrocele serum, one part, and plain agar condensed to two-thirds its bulk, two parts; with an acidity to phenol phthalein of 1 per cent. With a medium containing ascitic fluid in place of hydrocele fluid, isolation was not accomplished, but cultivation of the organism was equally successful, growth being supported over periods of from four to seven days."

Number of Cases.—The epidemic extended from August, 1902, to September, 1903. During this period 319 children were admitted to the hospital, exclusive of the maternity and scarlet fever annexes, the average age being seven years and two months. There were 151 girls and 168 boys. The cases known to be infected numbered 19, only 1 of these being a male.

Age.—The extremes of ages were six months and thirteen years; between these there were one of eight months, two of two

years, seven of three years, two of four years, three of six years, one of nine and one-half and one of ten years, respectively. Seven of these cases were three years old and but 3 of the 19 cases were over six years of age.

Chronology.—The first appearance of this infection in the children's ward was on August 8, 1902. No other child developed the disease until October 10th. After this 5 cases appeared on the 23d, 25th, 26th and the 31st. No more gonorrhea was reported for nearly two months, or until December 24th, another following quickly on the 26th, with two more on the 7th and 11th of January, 1903. After this no new cases appeared in the hospital till April, when three infected girls entered from the scarlet fever annex. The next vulvovaginal discharge was reported on June 3d, two on the 8th and one on the 12th, which completed the series.

Duration.—The record of the cured cases shows the average duration to have been 116 days; the shortest period of discharge was 40 days; the longest, including exacerbations and remissions, was 252 days.

Several of these patients showed apparent recoveries with negative smears, in whom the discharge appeared later.

Physical Condition of Patients.—Of interest for its possible influence on the duration of the infection, the average of which seems to be longer than is usually recorded, would be a consideration of the physical condition of the patients at the time of gonorrheal infection. Five of these patients were infected during the course of or while convalescent from typhoid fever and five were tuberculous; four had scarlet fever at some period of the disease; three had been operated on for cleft palate; two with congenital dislocation of hip had been treated by the Lorenz method; one was epileptic; one followed operation for genu valgum; one for anal fistula and one for venous angioma. The apparent lowered vital conditions in the majority of these cases cannot be overlooked in considering the persistence of the infection.

Complications.—Of these patients four received treatment for conjunctivitis, three of which were of very mild type. The fourth, the only one in which the discharge from the eye was examined, showed streptococci only. This case of ophthalmia was quite obstinate, receiving 10 per cent. protargol applications daily for a week. In this case the eye affection developed two days before the vaginitis.

One baby of six months developed a mild conjunctivitis probably four or five days after the vaginitis. A third ophthalmia of a mild type appeared in a six-year-old three days after a negative test of the vaginal secretions. The fourth, an eight-months' baby, had a slight eye discharge nine days prior to the vulvovaginitis.

Five cases were reported with suppurating ears. One, a six-year-old, had a double otitis following measles seventeen days prior to the vaginitis. One, three years old, had a right ear discharge following a pharyngitis, which occurred twenty-five days after the beginning of the vulvovaginitis. A three-year-old typhoid convalescent showed enlarged cervical glands, followed the next day by discharge from the left ear, on the thirty-third day of the infection. Number 4, another convalescent from typhoid, a child of four years, showed double otitis media a week following the vaginal infection. The 5th case developed ear trouble simultaneously with a scarlet fever eruption which preceded by nineteen days the vulvovaginitis. As no examination of the discharge from the ears was made and otitis media is known to be so prevalent among children, it would seem like straining a point to claim any etiologic responsibility to the gonococcus in these cases. These nineteen patients presented but one abscess, a suppurating axillary gland in a six-year-old typhoid convalescent, which was opened thirteen days before the vaginal discharge appeared.

None of these patients complained of articular pains, with the exception of a ten-year-old girl, who developed temperature and pains in both elbows and knees on the first day of a vaginal discharge, which occurred during convalescence from scarlet fever. This patient, also, furnished the only instance of peritonitis. Contrary to the observations of some, the vaginal discharge did not diminish with the appearance of peritoneal symptoms, nor was the onset severe. She made a good recovery from both vaginitis and peritonitis, and left the hospital a month after the development of the complication.

Intercurrent Infections.—Of intercurrent infections there were 4 of scarlet fever, 2 of measles and 1 each of pharyngitis, bronchitis and meningitis.

Sources of Infection.—The question of the sources of this infection requires a review of the chronology of its appearance and also the environment of the patients affected. The first discharge, observed August 8, 1902, was from the urethra of a two-year-old

boy under treatment for rectal fistula, seventeen days after his first operation, during which interim he had been taken daily to dressing room or clinic. The urethral secretion contained gonococci, but no microscopic examination of the discharge from the rectal fistula is reported.

Gonococci were next found in a vulvovaginal discharge in a typhoid fever patient two days after admission and five days after the reported cure of the first case. These two patients appear to have furnished the infection for this entire epidemic. Is it probable that this vulvovaginitis, which was noted forty-eight hours after admission, bears any relation to the gonorrhea of the boy, reported cured five days before? Both were diapered cases and it is barely within the possibilities that the little girl may have been exposed through an imperfectly disinfected napkin. Her sister, admitted the same day with typhoid, did not show a discharge until the eighteenth day of residence, 2 other cases having developed meanwhile. This long interval would appear to argue against the supposition that these sisters were infected at home. Six girls developed vulvovaginitis in the period of twenty-one days; 3 were typhoids and 3 were surgical cases; all were diapered and all had temperatures taken daily per rectum, with the ward thermometers. These six girls form a clinical group as no new cases appeared for *fifty-four days.* They were removed from the ward on the 4th of November, 1902, for the purpose of isolation, Room No. 301 being appropriated for their use. No infection occurred outside of this group until December 24th, when a vaginal discharge was observed in an eight-months-old surgical case. She had been operated for venous angioma of the face and was taken to clinic and dressing-room repeatedly. Search for the source of this infection brought to light the fact that her six-months-old cribmate, who showed a vaginal discharge two days later, had attracted attention by a suspicious odor for several days. A vaginal discharge from this baby may have been obscured by her profuse diarrhea. Further inquiry shows that this baby entered the ward December 12, 1902, from a room on the third floor adjoining that containing the six infected girls. She had been waiting there for an operation for cleft palate, which was performed December 13th, the day following her admission to ward. Is it possible that this baby was infected on the third floor while in proximity to the gonorrheal isolation ward? As the general children's ward was now quarantined for measles, these

two infected babies were retained, with the result that a vaginal discharge appeared in a four-year-old typhoid convalescent twelve days later, to be followed by another, January 11, 1903, after an interval of four days, *also* in a typhoid convalescent of six years. Both were diapered cases. No new patients being admitted, there remained in the ward four infected and two non-infected girls and five or six boys until February 14, 1903, when the quarantine for measles was raised and the ward was fumigated. The eight-months-old girl was taken home; the 3 remaining cases were sent to an isolation ward on the sixth floor, where they were soon joined by the four infected girls remaining from the first group, two having left the hospital. The six-months' baby died two days later, February 16th, of pneumonia, following the measles.

It is of interest to note here that a six-year-old girl, admitted to children's ward November 24, 1902, and operated on for cleft palate, is reported by the mother as showing a vaginal discharge the day after reaching home. She left the hospital December 12, 1902, no vaginal discharge having appeared at that time.

The next 3 cases form a group in which this infection was first observed at the scarlet fever annex, to which they had been removed from the children's ward. They were all surgical cases, who showed scarlet fever on the 28th of February, 2d and 6th of March, respectively. The discharge was first observed in the last patient to go, a two-year-old cleft palate case, who was reported with vaginitis the day she was transferred, although none had been discovered before leaving the children's ward. Four days later, March 10th, a four-year-old girl of the second gonorrheal group, isolated now on the sixth floor, developed scarlet fever and was transferred to the annex the same day, March 10, 1903. The annex records show that the discharge appeared in the other 2 cases on the 19th and 21st of the same month.

After the subsidence of the scarlet fever on the 6th of April, three of these patients were sent to the isolation ward on the sixth floor, the fourth having been taken home.

The last group comprises 4 surgical cases. The first, a three-year-old tubular spondilitic, admitted April 16th, showed vaginitis June 3d. The second, a thirteen-year-old, admitted April 22d, was operated May 25th for tubercular glands of the neck, and developed gonorrhea June 12th. The third, a three-year-old,

entered May 11th for correction of congenital dislocation of hip, and developed vulvovaginitis June 8th. The last, another three-year-old, admitted May 13th, with tubercular joint lesions, was operated May 19th, and developed gonorrhea June 8th.

Treatment.—It was found that the discharge yielded only to the more heroic forms of treatment. Most of the cured cases required 10 per cent. protargol douching every four to six hours, preceded by 1:2000 permanganate potassium solution, some obstinate cases requiring additional packing of the vagina with gauze saturated with 2 per cent. protargol.

Condition on Leaving the Hospital.—Of the 18 patients who showed the infection in the hospital, 10 were discharged cured, 2 died (intercurrent disorders) and 6 were removed by their friends while still under treatment. Those patients were pronounced cured from whom repeated smears were negative for nine days after cessation of treatment.

CONCLUSIONS.

Every hospital for children must be protected against infection from without by well arranged detention wards, in which the newly admitted patient may be submitted to the closest scrutiny for a period of at least fourteen days, to determine his freedom from acute infections before admission to the general ward.

A complete hospital should have ready for use, properly located isolation wards to which patients may be removed upon the first appearance of suspicious symptoms of an acute infection.

Gonorrhea is a most formidable disease in a children's hospital, and female infants are peculiarly susceptible to this disease.

A patient in a children's hospital showing gonorrhea should be promptly isolated and placed in charge of a special nurse. The interne or nurse while treating a gonorrheal patient should be relieved from other duties in the children's ward. The efficiency of any isolation may be questioned which does not individualize strictly not only in regard to the treatment, care and manipulation of the patient, but also as to the use of clothing, bed linen, feeding utensils, thermometers, douche pans, etc.

The same care by nurse and physician in sterilization is necessary as would be observed in going from a child with any acute infection to a non-infected child.

The question of exacerbation or reinfection as the cause of

reappearance of the gonococci in the secretions after repeated negative tests is an interesting one. This is especially true, where more than one infected child is isolated in the same room with the preponderance of probabilities in favor of the reinfection.

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DISCUSSION.

DR. NORTHRUP.—Every one of us has noted the wonderful susceptibility of the vagina and vulva to this infection. It is interesting that we seldom see the eyes affected when the vagina is so easily and persistently affected.

DR. KERLEY.—What has Dr. Cotton's experience been with gonorrheal arthritis?

DR. COTTON.—In the series of 19 cases reported there was but 1 case of joint involvement, and that followed a scarlet fever infection. I have had but a limited experience with gonorrheal arthritis in children. I recall but 1 case in which the arthritis was gonorrheal, and this occurred in a little girl, six years old, who contracted the gonorrhea from her father.

In the series there was but 1 severe case of conjunctivitis and 4 cases of slight conjunctival disturbances. The cultures did not show the organisms.

In regard to the greater susceptibility of the vagina and vulva over the conjunctiva in children, I believe this is due to the fact of greater exposure; children not diapered usually escape, while those diapered contract the disease more frequently. Again, the possibility of carrying infection by means of thermometers should not be forgotten. I believe this to be a fruitful source of the disease in very young children.

DR. JACOBI.—I have been struck with the fact that when the disease is located in the vagina it is rarely found in the conjunctiva, while, really, one would expect the reverse to take place. Children who are not diapered make their fingers go everywhere, and, by preference, about the abdomen; they must be expected to carry pus to the eyelids, and yet this rarely happens. But what does happen is a communication from one patient to another, until a whole ward becomes affected.

All have seen secondary infections from the cervix to the uterus and occasionally a nephritis and peritonitis; I believe all of us have had such cases—some very bad; such cases run a more favorable course than in adults. Dr. Huber drew attention to this in a paper some years ago. Cases of peritonitis secondary to gonorrhea are apt to get well. Dr. Northrup will well remem-

ber 2 cases occurring in the same family; in one a very severe arthritis developed, but the child finally got well.

I have seen 1 case lately which occurred in an unusual way. The baby was newly born. The mother died of puerperal sepsis on the fourth day after her confinement. On the eighth day the baby had a conjunctivitis and was brought into the ward. This was found to be a gonorrheal infection. The eye got better, the other eye was protected and did not become affected. About the fourth or fifth day (the child was eleven days old) the left side of the neck became tumefied, the glands became swollen, abscesses formed, were opened and found to contain gonococci. At the same time the left hand became swollen, abscesses formed, were opened and found to contain gonococci. The opening was made in the palm of the hand. The baby died on the sixteenth day. This child became infected in its passage through the vagina of the mother. The difficulty of curing these cases is great; there is an anatomical reason for this. There is a very narrow introitus. It has been recommended that the hymen should be removed, but this is a cruel procedure to which few will stoop. The difficulty of cleansing the parts is very great. Great difficulty is experienced in cleansing the cervix; the disease ascends to the uterus; between the third and fifth year the uterus grows only in its cervix. The inside of the organ soon becomes infected. The mucous membrane of the uterus is in folds and remains so until the ninth, tenth or eleventh year. It is between these folds that the gonococci find an abiding hiding-place, which resists treatment much more obstinately than even the posterior part of the male urethra. This is the reason for the difficulty encountered in reaching and attacking these organisms and explains, also, why the disease breaks out again when apparently cured. I have seen a number of cases during the past two years, in which the disease first appeared after the child had been in the hospital five, six, seven or eight weeks. I remember 1 case, which appeared when there was no other case in the ward; the child evidently came in with the disease and nobody noticed it. The disease may remain undiscovered for many weeks and months, just as in the male urethra. I know of nothing so difficult of removal as gonococci in the vagina and uterus.

DR. KERLEY.—I have an interesting observation to report, which was made at the Babies' Hospital during the service of Doctors Holt and Kimball. There have been as many as 18 to 20 cases of gonorrheal arthritis there during the past two years. It was peculiar in that, in the majority of the patients, who were boys, the infection took place without any external manifestation of the infection. Dr. Kimball has reported 8 cases, and of these, 7 took place in boy babies; the only possible source of infection was in the mouth through a stomatitis.

DR. LA FÉTRA.—I have seen several of the gonorrheal arthritis

cases at the Babies' Hospital during the past year, and within the past three weeks I have had 2 additional cases of this disease at the Vanderbilt Clinic, one in a baby three weeks old, the other in a child of eight months. The site of infection in the smaller baby was a circumcision wound, the history being given that the mother had had gonorrhea at the time the baby was born. There was no ophthalmia, but a balanitis. The ankles, wrists and interphalangeal joints were involved. The point of invasion in the larger baby was probably a granulating umbilical stump.

These cases of gonorrheal arthritis are many of them really cases of periarthritis, the exudate being in the tissues outside the joint. The remarkable thing about this type of arthritis is that most of the cases do well with nonoperative treatment, such as wet dressings of alcohol, or of ichthyol, along with immobilization of the joints.

DR. ACKER.—I have seen but 2 cases that were complicated. One of these complications occurred in a girl, a cystitis; the other was an arthritis.

I find that, as a rule, the cases yield readily to treatment with 25 per cent. ichthyol and glycerine, packed in the vagina.

DR. COTTON.—The treatment I employed was copious douching with permanganate of potassium solution, 1-2000, followed by 10 per cent. protargol. In at least 6 of the cases the vaginas were packed with 2 per cent. protargol gauze, applied with dressing forceps. As I recall it, this was more efficient than anything else used. It has occurred to me that it would have been better to treat the cervical canal itself, because this canal is usually invaded by the organisms. If this were done it might be possible to get rid of the disease. How is it possible, otherwise, to get at the microorganisms after they have taken up their residence in the submucosa of the cervical canal?

DR. CAILLÉ.—Some years ago the attention of the Post Graduate Hospital was called to these cases because of a suit against the institution. A child was admitted apparently in good health and without noticeable vaginal discharge; within ten days she developed a severe specific vulvovaginal infection. Before admission this child had been examined most carefully, but nothing was found. Now, before admission, these children are most carefully examined and swab cultures made; if any specific condition is found they are rejected; if not found they are admitted. In this way we are able to keep out all cases of vulvovaginitis, although occasionally a case will develop in the hospital.

In the treatment of such cases the best results are obtained by swabbing the parts with a nitrate of silver solution, 5-10 per cent. protargol, and with permanganate of potassium. We never used the fountain syringe, as we have seen injury result from the use of the douche.

DR. ADAMS.—Usually these cases are admitted to the hospitals devoted to the care of children; if such cases are excluded because they possess some degree of contagion, what would become of these hospitals for children? In the Children's Hospitals in Washington, D. C., we admit these cases of vulvovaginitis whether they are specific or not. So far we have not felt the necessity for excluding them.

Recently I have seen 7 cases of vulvovaginitis in the same family, undoubtedly specific. Direct infection was unquestionable, the question of the infection rested between the father and the mother; both undoubtedly had gonorrhea, and the mother claimed that she contracted the disease from the father. These children had slept alternately in the bed with their parents. All the children were within a short time admitted to the institution and treated successfully. So far as the babies' wards are concerned, we have a limited number of beds, only twelve, and the cases are not selected ones: occasionally we receive such an infectious case; when we do the doctors and nurses are impressed with the fact that the treatment of the case and its cure are not so important as the prevention of infection of other children in the wards. I do not know whether the experience in the Foundling Asylum agrees with my own or not. During six months there are usually entered about fifty babies, and they are brought from all the walks of life, the well-to-do and the vicious classes. In ten years I have yet to see a case of gonorrheal vulvovaginitis in that institution. We do have cases of conjunctivitis in the maternity service of hospitals sending babies to this institution; but investigation does not show always that they are of a specific nature. I believe that these cases, especially the girls, should be carefully examined before admission to the institution. Cases received in Washington conceal many pathological germs which later manifest themselves; they are latent and may become active later on. The nurses should be educated regarding the handling of these infectious cases and all precautions taken to prevent their spread; they should be taught the rules regarding cleanliness; if such education was enforced much of the difficulty would be overcome. I believe it is far better to admit these cases than to turn them away and let them depend upon the dispensary for their treatment. Judicious, systematic and symptomatic treatment should be employed.

PERICARDITIS (GONORRHEAL).

BY F. HUBER, M.D.,
New York.

The old view that gonorrhea is simply a local disease has been discarded for many years. General infection of gonorrheal origin and the various metastatic processes, which may arise in consequence, have been carefully investigated during the past decade. The joints, bones, tendons and bursae, it is well known, are frequently implicated. Even the nervous system does not escape the inroads of the germ. In fact, all the different structures of the body may be involved. In some instances the lesions are due to action of the specific microorganism, in others the toxins cause the trouble. Now and then both may act.

A closer study of this part of the subject, as shown by a review of the literature, is attended with many difficulties. From the nature of the case experiments upon man cannot be carried out and the lower animals seem to be immune. Gonococci may reach distant organs or structures in two ways. (*a*) Through the circulation. (*b*) By means of the lymphatics. Up to the present it has not always been possible to assert positively that certain lesions are due to or can be directly attributed to the action of the toxins or to the germs themselves.

In general it is believed that arthritic processes, endo-, myo- and pericardial affections, pleurisy, periostitis and phlebitis are caused by the gonococcus, the majority of the others by the toxins. It should not be forgotten, however, that in many instances a mixed infection has occurred.

Lesions of the deeper portion of the genital tract, a lowered condition of the constitution, cachetic states, serious constitutional disturbances and meddlesome instrumental interferences are some of the more important factors favoring general infection. Not only is there a difference in the virulence of the microorganism, but there is also a variation in the susceptibility of the subject. When the anterior parts are involved, the toxic products are readily washed away. In the posterior urethra and deeper structures on the contrary, the crypts and glands unfortunately permit the offending secretions to remain in the recesses. Clinically, it has long been recognized that untoward results are less apt to follow in cases in which the anterior urethra only is involved. General infections resulting from gonorrheal affections of the vulvo-

vaginal tract, rectum or conjunctival mucous membrane, are common. Kimball, however, reports a series of cases in infants, in whom *no local lesions* could be found to explain the mode of entrance of the organism. The suggestion is made that from a stomatitis due to the gonococcus such a systemic infection may arise.

Elevations of the temperature, more or less malaise, loss of appetite, etc., are not uncommonly observed at the outset. These symptoms constitute the early manifestations of a systemic infection.

The skin does not escape—erythema, urticaria, erythema nodosum, hemorrhages, bullous erythema and hyperkeratosis have been reported. Muscles and connective tissue at times show the effects of the malign influences of the microorganism.

The invasion of lymphatics, inguinal adenitis, terminating in resolution or suppuration, is an old story. The danger of an extension into the urethra, bladder, ureters and even to the kidneys is well known. Quite recently it has been shown that toxic effects involving the kidneys may occur as a metastatic process without upward extension or a direct involvement of the bladder or ureters. It is generally conceded that in females of tender years, and even in infants, the uterus, tubes, ovaries and peritoneum (local or general) may be implicated.

Septic and pyemic processes occur with more or less frequency in childhood. Ophthalmia from direct infection by the fingers, is unfortunately quite common. Abscesses in the subcutaneous tissues and muscles have been reported within the last few years. Involvement of one or more joints, tendons, etc., may occur in infants within the first few weeks of life. Serous membranes, particularly the endocardium, may be implicated. The process is met with in all grades from the simple to the ulcerative variety.

The writer is indebted to the late Dr. J. Berenson for the opportunity of studying the following interesting case:

Jenny A., three and one-half years old, born in New York of Roumanian parentage, enjoyed excellent health until the present illness. She was taken acutely ill with swollen ankle, attributed by the mother to infection from a scratch with a piece of tin. Upon closer examination the accident was found to have occurred in the opposite limb. Temperature 104°F., pulse accelerated. The joint became more swollen and tender without any particular redness. The opposite knee became painful, and the

patient sweat considerably. On the third day, pericardial friction sounds, with a loud systolic murmur over the precordial region, were detected.

As the general surroundings were bad the child was sent to the hospital June 3d.

On Admission.—Stout, well-nourished child, color good. Lips dry, tongue moderately coated. Slight general redness of throat. Abdomen normal: spleen not palpable. There was slight edema of the feet. *Heart.*—Apex, impulse well defined in fifth space in nipple line three inches from mid-sternum. Cardiac dullness begins to right of sternum: action regular and rapid. To left of sternum and at apex a loud blowing systolic murmur is heard. Over the precordial region numerous superficial to and fro friction sounds are audible, intensified by inspiration and heard in expiration as well, though less distinctly. Second pulmonary sound somewhat accentuated.

Temperature 102°F., pulse 133, irregular in force and frequency, respiration 38. The bowels were constipated. No history of sore throat. Difficulty in breathing and severe precordial pains were the principal symptoms complained of. Urine, amber, no sediment; specific gravity, 1.030; albumin, faint trace. There was an excessive amount of indican; leukocytes from vulvovaginal catarrh.

Under the impression that the trouble was of a rheumatic nature, antirheumatic measures were resorted to, without any material benefit. Ice bags were placed over the heart. Fluid diet ordered. From June 5th to 8th vomiting occurred, the pulse became irregular and somewhat slower. The vomiting was incessant and not readily controlled; nutrient enema were ordered. From eighth day on the stomach was under control. Child apathetic. Soon after this the child gradually improved. An attack of urticaria occurred about the 18th day, which we could not attribute to the diet, as milk and cereal decoctions only were allowed. From the nineteenth day on the pulse became slower. The vaginal discharge showing gonococci became more profuse. Local treatment could not be carried out because of the struggles and excited conditions of the patient. General supporting treatment only was possible.

The temperature 102°F. on admission gradually dropped to 98°F. on the third day, then ran between 98°F. and 99°F. for three days. For the next few days it hovered in the neighborhood of 100°F. On the thirteenth day it rose to 102°F., followed by a

rather abrupt fall to 98°F., and continued to make short excursions between 98°F. and 100°F. for the next two weeks.

The pulse rate was high in the beginning; in a short time it came down and ranged from 92 to 100 for some time. In the third week it became slower and was often intermittent in character, varying from 60 to 84 at different times. In the early part of July it ranged between 80 and 100 per minute.

The pericardial friction sounds continued for several weeks. The murmur was irregular in its distribution depending upon the heart's action. No signs of effusion could be detected by percussion. About the beginning of July the murmur was very faint. At the time of discharge the sounds were normal. Heart's action good, no hypertrophy or abnormality was appreciated. At a subsequent examination, several months later, there were no evidences of any change in the heart's action. The sounds were normal and the physical condition excellent.

The first diagnosis of acute rheumatism was questioned early in the course of the disease. The prominent swelling of the joint, the subsequent progress of the case, the marked evidence of a low grade of sepsis, the projectile vomiting, the variable character of the murmur and its complete disappearance without appreciable change in the valves, lead to a probable diagnosis of infection due to the gonococcus—fortunately of a mild and not ulcerative type, confined to the pericardium and myocardium.

The gonorrheal infection may affect the endo-, myo- and pericardial structures. These affections are frequently combined. The endocardium is most often involved. The inflammation may occur as a simple process or show itself as an ulcerative type, with fatal termination. Myocarditis due to the specific cause under consideration is rare; it is usually secondary to the endocarditis.

Pericarditis, though somewhat frequent, is rarely the only manifestation. The affection is generally benign, does not give rise to much discomfort and may run its course without any manifest symptoms.

Objectively, it may occur as a dry form or may be attended with exudation. In the first variety the pericardial friction sounds are distinctly heard, in the other case dullness exists with feeble heart sounds. The effusion is generally serous in character, at times though it may be hemorrhagic. Beurmann has reported a case in which pus was formed. Other instances are mentioned in which several serous surfaces were involved (meninges, pleura, etc.).

ACUTE LEUKEMIA IN EARLY LIFE.

BY FRANK SPOONER CHURCHILL, M.D.,
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Acute leukemia is a comparatively rare disease in early as well as in adult life, but with modern methods in blood examinations cases are now recognized more frequently than formerly. Morse,[1] in 1898, reported a case in a four-year-old girl and gave a brief *résumé* of 7 other cases under ten years of age collected from the literature. McCrae,[2] in 1900, reported a three-year-old boy with leukemia, giving abstracts of 13 cases, including Morse's. Since McCrae's article I have collected 14 cases, brief abstracts of which follow:

CASE I. (Strauss.[3])—Boy, aged thirteen months; family and personal history good. Illness began two or three weeks ago, with enlargement of the abdomen. General condition good; no digestive disturbance; no nose-bleed. Physical examination showed marked pallor, slight rickets, numerous petechiæ, teeth good, tonsils normal, chest normal, spleen much enlarged, urine negative. Continued thus for two or three days, then became worse, and died three weeks from onset.

Blood.—Hemoglobin, 25 per cent.; erythrocytes, 2,008,000; leukocytes, 208,000; lymphocytes, 97.3 per cent.; large mononuclears, 0.6 per cent.; lmphocytes varying in size. Some poikilocytosis and polychromatophilia; few normoblasts.

CASE II. (Bloch and Hirschfeld.[4])—Boy, aged eight months; family and personal history unknown. For a few days has been unusually sleepy; no other symptoms. Physical examination showed marked anemia, right-sided pneumonia, lymph glands and spleen enlarged, rickets. Under observation for a few days, then had double pneumonia, and died.

Blood.—No total white count given, but proportion of whites to reds was 1 to 12. Polynuclears, 28 per cent.; myelocytes, 10.2 per cent.; small mononuclears, 27.14 per cent.; large mononuclears, 38.8 per cent.; poikilocytosis, megaloblasts, and normoblasts; eosinophiles and mast-cells rare. Duration unknown, but probably acute. Regarded as lymphatic myelogenous type;

autopsy showed leukemic changes, especially interesting on account of changes in central nervous system.

CASE III. (Janeway.[5])—Boy, aged six years. Trouble began three months ago with swelling over masseters, later swelling of cervical and other glands, and marked pallor; no hemorrhage and no retinitis.

Blood.—Hemoglobin, 40 per cent.; leukocytes, 219,000; large lymphocytes, 87 per cent.; small lymphocytes, 11 per cent. Clinically, the case was chronic, but the blood was that of the acute type, and the author cites the case to show the impossibility of regarding acute lymphatic leukemia as a separate disease.

CASE IV. (Jewett.[6])—Boy, aged thirteen months, smaller and weaker of twins. Family history negative. Personal history: never robust.

March, 1900. Pale, occasional diarrhea, malnutrition. April: Slight, irregular fever. May: Tumor in left abdomen. Never any enlargement of superficial lymph glands; never any hemorrhages.

Examination.—June: Moderate emaciation, pale, spleen much enlarged, reaching to level of umbilicus.

Blood.—Hemoglobin, 30 per cent.; erythrocytes, 1,912,000; leukocytes, 21,000; lymphocytes, 87 per cent.; neutrophiles, 10.5 per cent.; eosinophiles, 1.5 per cent.; myelocytes, 1 per cent.; eight normoblasts and eight megaloblasts in 200 cells; lymphocytes of all sizes; poikilocytosis and polychromatophilia; fever marked; regarded as subacute lymphatic leukemia. Death, June 17th. No autopsy.

CASE V. (Jewett.[6])—Girl, aged seven months. Family history negative. For three months pallor, enlarged spleen, emaciation, diarrhea, irregular fever, great weakness, enlarged glands. No hemorrhages.

Blood.—Hemoglobin, 20 per cent.; erythrocytes, 1,150,000; leukocytes, 33,000; lymphocytes, large and small, 78.6 per cent.; neutrophiles, 18.2 per cent.; eosinophiles, 1.8 per cent.; myelocytes, 1.4 per cent.; fourteen normoblasts in 500 cells. Death at eight months. Diagnosis: subacute lymphatic leukemia. No autopsy.

CASE VI. (Pollman.[7])—Girl, newly born. Family history negative. Mother's blood normal. Sickly at birth, never cried nor nursed, apathetic, petchiæ on forehead and lower limbs, later universal. At sixteen days cyanosis; liver and spleen much enlarged;

cervical and inguinal glands somewhat enlarged; temperature from 38.8° to 41.5° C. Death at nineteen days.

Blood.—Erythrocytes, 2,500,000; leukocytes, 312,500. No differential count, but leukocytes, almost all mononuclear, with rather a large nucleus. Autopsy: great enlargement of spleen and liver and thymus, *open ductus arteriosus.* Tricuspid endocarditis. Culture from spleen negative. Culture from heart blood showed only colony of undetermined organisms. General infiltration of tissues with leukocytes. No examination of bone-marrow. Regarded as a case of splenomedullary leukemia, but probably was lymphocytic.

CASE VII. (Stewart and Campbell.[8])—Boy, aged six years. Family history negative. Caught cold in March, 1901, never well afterward. Pallor, poor appetite.

Examination.—April, 1901. Pale, submaxillary glands enlarged. Seen several times during April and May. Irregular temperature, nose-bleed, pain in legs. Spleen not enlarged.

Blood, May 26, 1901. Hemoglobin, 21 per cent.; erythrocytes, 750,000. After large hemorrhages: leukocytes, 40,000; lymphocytes, 25 per cent.; polynuclears, 45 per cent.; myelocytes, 27 per cent.; eosinophiles, 3 per cent.; normoblasts fairly numerous. Death two days later.

CASE VIII. (Einhorn.[9])—Girl, aged nine years. Family history not mentioned. Well until several months ago, then weakness, later diarrhea.

Examination.—Not pale, cervical and inguinal glands enlarged, spleen and liver enlarged, chest negative.

Blood.—Erythrocytes, 3,360,000; leukocytes, 389,000; small lymphocytes, 48.7 per cent.; large lymphocytes, 49 per cent.; mononuclears, 1.3 per cent.; polynuclears, 1 per cent. No further history given.

CASE IX. (Rocaz.[10])—Boy, aged four years. Came to hospital with tonsillitis, no diphtheria bacilli. Family history good. Previous history: subject to sore-throat. May 25, 1901, ill three days with tonsillitis, which subsided, but temperature remained high. In two days fetid diarrhea.

Examination.—Chest normal, tonsils enlarged, spleen enlarged. Widal reaction positive; case therefore regarded as typhoid. Four days later cervical and inguinal glands enlarged.

Blood.—Hemoglobin, 30 per cent.; erythrocytes, 1,816,000; leukocytes, 244,000; lymphocytes, 96.6 per cent.; large mononu-

clears, 0.6 per cent.; transitional, 0.6 per cent.; polynuclears, 2.4 per cent. Some poikilocytosis, later petechiæ, bleeding of gums. Death twenty-seven days after onset. Autopsy: hypertrophied thymus, infiltration of tissues generally with small cells.

CASE X. (Fowler.[11])—Girl, aged five years. Family history good. Previous history: measles, whooping-cough, chicken-pox Two months ago sore throat, enlargement of cervical glands, pale, no appetite, no pain, weak. One month, hard swelling in abdomen.

Examination.—Pale; tonsils enlarged, mouth breather. Chest: soft bruit at apex of heart; superficial glands slightly enlarged, spleen and liver much enlarged; retinitis (right); urine negative. Never any hemorrhages; temperature elevated.

Blood.—Erythrocytes, 2,700,000; leukocytes, 240,000 to 250,000; small lymphocytes, 7 to 13 per cent.; large lymphocytes, 32 to 56 per cent.; polynuclears, 16 to 22 per cent.; myelocytes, 12.1 to 27 per cent.; eosinophiles, 0.6 to 2.6 per cent.; eosinophilic myelocytes, 0.7 to 3.4 per cent.; mast-cells, 0.3 to 4.4 per cent.; normoblasts numerous. Regarded as intermediate between lymphatic and myelogenous leukemia. Died at four and one-half months after onset.

CASE XI. (McCaw.[12])—Infant, aged twenty months. Family and personal history good. Three weeks ago began to walk feebly and stagger, drowsy, and apathetic. Two weeks ago swelling of glands in neck, enlargement of abdomen.

Examination.—Not pale, though hemoglobin 38 per cent. Glands in neck, axillæ, and groin enlarged; spleen much enlarged; liver enlarged. Urine: albumin, oxalic acid crystals.

Blood.—Leukocytes, 810,000; lymphocytes, 99.2 per cent.; later petechiæ and once blood in bowel movement. Spleen and glands diminished in size. Died about six weeks after onset.

CASE XII. (Taylor.[13])—Boy, aged ten years. Six weeks ago pain in legs, thirsty, yawning, enlargement of parotid and submaxillary glands, of liver, and left kidney.

Blood.—Leukocytes, 15,000; lymphocytes, 46 per cent. A few days later leukocytes, 50,000; lymphocytes, 94 per cent., with increase in size of glands. Hemorrhage from mucous membranes. Died twelve weeks after onset. Autopsy showed general lymphatic infiltration.

CASE XIII. (Geissler and Japha.[14])—Boy, aged five years. Family history negative. Previous history, chicken-pox. Two months before became ill with fever, pain and swelling of abdo-

men, pain in feet, and later in all joints, general weakness; symptoms persistent ever since.

Examination.—Well nourished, no rickets, chest negative, abdomen negative; urine negative; no swelling of joints nor pain on movement; no exanthem; slight enlargement of submaxillary and inguinal glands.

Blood.—Hemoglobin, 55 per cent.; erythrocytes, 2,200,000; leukocytes, 18,800; lymphocytes, 80 per cent. A few normoblasts, no poikilocytosis. Later petechiæ, nose-bleed, great enlargement of spleen and liver, no glandular swelling, increasing bone pains. Blood during the next three months ranged as follows: hemoglobin, 22 to 60 per cent.; erythrocytes, 158,000 (after a nose-bleed) to 2,208,000; leukocytes, 12,000 to 34,150; polynuclears, 3 to 24 per cent.; mononuclears, 76 to 97 per cent. Poikilocytosis, many megaloblasts temporarily after severe hemorrhages, many megalocytes in groups and about four times as large as a red corpuscle. A few neutrophilic and eosinophilic mastzellen, polychromatophilia. Death about five months after onset. Case at first might have been regarded as one of subacute rheumatism had it not been for the blood examination.

CASE XIV. (Glinski.[15])—Boy, aged one year. No family history. No previous history. For indefinite time pallor, malnutrition, vomiting and diarrhea, prominence of eyes.

Examination.—Pale, poorly nourished, submaxillary and cervical glands enlarged; bronchopneumonia on both sides; heart weak; spleen enlarged and hard; liver not enlarged; marked exophthalmus.

Blood.—Hemoglobin, 21 per cent.; erythrocytes, 918,750; leukocytes, 180,416; lymphocytes, 94.4 per cent.; neutrophiles, 5.3 per cent.; a few myelocytes and nucleated reds. Died with symptoms of pneumonia and heart failure. Autopsy, widespread leukemic changes, especially in pia mater.

There can be no doubt of the diagnosis of leukemia in any of the above cases. In all but Cases II., IV. and V. the blood count settles it beyond question. In Case II. there is no doubt of a marked leukocytosis, even though no total white count is given, the proportion of white to red being 1 to 12. The differential count was probably modified by the pneumonia, for we find 28.8 per cent. of polymorphonuclears, a high proportion for leukemia. The total mononuclears, 60 per cent., would not be high for an eight-months infant in health, but is rather high for a leukemia

with 10 per cent. of myelocytes, and, together with this percentage of the last-named cell, justifies classing the case as one of the mixed-cell variety. Finally, the diagnosis is clinched by finding at autopsy typical leukemic changes. As to Cases IV. and V., the advocates of von Jaksch's anemia as a distinct disease might possibly class them as such; but I cannot recognize that anemia as a distinct and separate disease, as a primary anemia. It is secondary, the result of a variety of causes, chief among which are rachitis, syphilis, and severe intestinal disturbances. There is nothing in the reports of these cases to lead one to class them as secondary anemia, while the leukocyte counts of 21,000 and 33,000, with the lymphocyte counts of 87 and 78 per cent. justify the diagnosis of lymphocytic leukemia. That such comparatively low counts do occur in leukemia is evidenced by Case XII. above, where were found at first a total count of 15,000, a lymphocyte count of 46 per cent., which figures a few days later had increased to 50,000 and 94 per cent., respectively.

My own case, now to be given in detail, also shows how low the total count may go. The history of this case, attended by Dr John W. Fletcher, of Winnetka, and myself, is as follows:

September 1, 1903.—A. B., aged four years.

Family History.—Maternal great-grandmother and paternal grand aunt died of phthisis. Father had trouble with ankle in infancy, said to be tubercular; outgrew it apparently, but for two or three years before birth of patient had more trouble with same ankle, though an exact diagnosis was never made. Two brothers, aged seven and nine years respectively, are well, but not robust, the former having adenoids. Sister, two months old, apparently healthy.

Previous History.—Well at birth. Breast exclusively for three weeks; then breast and modified milk until seven or eight months, when weaned. General health has always been good, except that at times since early infancy the skin has shown marked pallor. An attack of acute tonsillitis in December, 1902. Has had none of the acute diseases of childhood. Appetite has been good, as a rule. Bowels generally regular, occasionally constipated.

Present Illness.—For two or three months patient has had little appetite, and has shown a gradually increasing waxy pallor. There have been absolutely no other symptoms. The diet has been deficient in all directions, though more especially in the matter of fats and proteids. He has been up and about, out of doors play-

ing, has slept well, has not been fretful, bowels have been regular. In a word, except for his loss of appetite and great pallor, he has seemed perfectly well.

I first saw him professionally on September 1, 1903, at which time the result of the physical examination was as follows:

Physical Examination.—Development and nourishment excellent, marked waxy pallor of skin and mucous membranes, shotty feel to cervical glands, both tonsils enlarged. Chest: area of cardiac dullness normal; apex, fourth space just inside mammillary line. Heart sounds strong and vigorous; soft systolic murmur heard generally over precordia, but loudest at the apex; no bruit in neck. Pulse 96, regular, good strength. Lungs negative. Abdomen negative. No signs of hemorrhage anywhere; no tenderness over long bones. Blood: erythrocytes, 3,500,000; leukocytes, 10,000; hemoglobin, 75 per cent. (Tallquist).

Directions were given to increase his diet in all directions, and he was given liquor potassi arsenitis in gradually increasing doses.

September 9th.—Urine, 400 cc.; specific gravity, 1021; no albumin, no sugar. Sediment: slight excess of leukocytes, a few renal cells, a very few normal blood corpuscles.

15th.—Eats more under compulsion, but is no better; weight, forty pounds. Urine: specific gravity, 1020; no albumin, no sugar. No excess of indican. Sediment as before.

17th.—Patient more easily tired, and is fretful. Physical examination: pallor is more marked, petechiæ present on neck, trunk, and limbs; not numerous; noticed by mother two days ago. Numerous black-and-blue spots in various places. No tenderness over the bones. Chest as before. Abdomen: spleen is felt one inch by one inch below the ribs. Liver not enlarged, no distention. Blood (see table). Diagnosis: lymphatic leukemia.

20th.—Seen in consultation with Dr. Frank Billings, who concurred in the physical examination as reported above and in the diagnosis. Blood (see table).

21st.—Ten cc. of blood withdrawn from the median basilic vein by Dr. Ludwig Hektoen, who subsequently reported presence of the streptococcus.

22d.—Sixty cc. of human blood serum withdrawn yesterday from a vigorous woman were injected into the patient at 9:30 A.M. At the suggestion of Dr. Fletcher x-ray exposures were begun at 5:30 P.M. under the direction of Dr. W. A. Pusey. Blood (see table). Temperature, same.

23d.—Much pain and discomfort from the serum injection. A few petechiæ. Blood (see table).

24th.—Temperature higher, otherwise condition the same. Daily salt enema begun.

25th.—Blood (see table).

26th.—Spleen not felt and apparently not enlarged on percussion. Pulse between 132 and 134, strong and regular during the day, irregular during the night. Heart action strong and regular; murmur more intense and heard everywhere over cardiac area; some twitching of limbs; involuntary bowel movement at 8 P.M. Patient is decidedly worse. Blood (see table).

27th.—Blood in bowel movements to-day for the first time; general condition same as yesterday.

28th.—Much worse this morning; at times irrational and mildly delirious. More blood in bowel movements; pulse weaker, respiration becoming irregular. Oxygen started at noon—ten minutes, every twenty minutes.

29th.—Moribund at 9 A.M. Unconscious and Cheyne-Stokes respiration at 10 A.M. Gradually sank, and died at 2 P.M.

Date.	Hemoglobin.	Red.	Leukocytes.	Mononuclears.	Polymorphonuclears.	Eosinophiles.
September 1, 1908..	75 %	3,500,000	10,000	.....	.....	.....
" 16, " ...	50 %	.....	Much increased.	99.5	0.5	.....
" 18, " ...	45 %	3,116,000	30,000	98.9	1.0	0.1
" 20, " ...	40 %	2,740,000	52,000	99.5	0.5	0.2
" 22, " ...	40 %	1,892,000	80,020			
" 23, " ...	.....	2,174,000	80,080	99.0	0.13	0.07
" 25, " ... 5 P.M.	25 %	1,516,000	20,040			
" 26, " ... 4.30 P.M.	20 %	1,544,000	6,300	98.2	1.55	0.3
				1000 cells 1 myelocyte?		1 normoblast.
" 28, " ... 4 P.M.	15 %	1,280,000	8,000			

This case presents some points of extreme interest. First as to his heredity, the question of tuberculosis must be considered. The history of this trouble, three generations back, can have but little bearing on the case. The exact character of the trouble in the father's ankle apparently never was determined. On the whole,

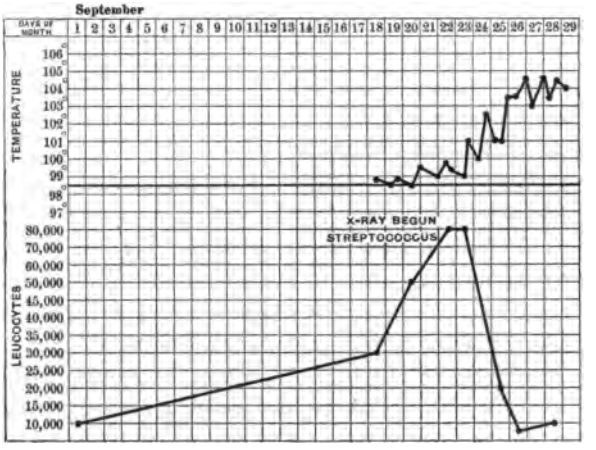
it is not probable that the tubercular taint played any part in the development of the child's final sickness.

When did this final trouble begin? This it is impossible to say. I saw the child frequently in a non-professional way throughout his life, and at times noticed his great pallor. Generally, however, he seemed perfectly well, so I said nothing to the family about his off days. His parents regarded him as the most robust one of three children. During the early part of last summer I noticed the increasing pallor, which was so great in early September that I then examined the child, with the result already detailed. There is no doubt in my mind that the malady began as early as last June or July, if not before. It is not at all improbable that the periods of intense pallor first noticed in infancy were in reality the beginning of his trouble. This, however, is pure speculation.

The absence on September 1st of all physical signs except the waxy pallor, enlarged tonsils, and condition of the blood is striking. The absence of hemorrhages until late in the disease is also quite remarkable. The presence of a few normal red blood corpuscles in the urine in the first examination may, of course, be regarded as a hemorrhage due to the leukemic condition. The corpuscles, however, were very few in number, not more than one often sees in the urine of less serious conditions. There never was any nose-bleed, never any bleeding from the gums, and not until September 15th, two weeks before death, were any hemorrhages noticed under the skin. Indeed, the general absence of symptoms, except the pallor, were frequently commented upon by the parents. It was not until the onset of the acute symptoms with rapidly rising temperature, about six or seven days before death, that the child seemed acutely ill. It is unfortunate that I have not a record of his temperature during the first two weeks of September, but as he was up and out doors running around, the temperature was not taken. The three days' normal temperature, when systematic readings were first instituted, makes it probable that up to that time the temperature had been normal. I can but believe that the last week of illness simply marked the final acute stage of a chronic process which began months, possibly two or three years, before.

The chief interest of the case, of course, centres in the condition of the blood, especially in the leukocytes. The course of the erythrocytes, as a whole, shows a steady downward trend throughout the period of observation. That of the leukocytes is most in-

teresting. (See chart.) Beginning with a normal count at the first examination, it rises rapidly during the first two weeks to 30,000, then still more rapidly, reaching its maximum (80,000) five days later, when after two days more we see an ante-mortem drop to a point even subnormal—6,300. The leukemic agent, whatever it is, apparently was in the ascendancy during the first three weeks, and as a result of its action upon the bone-marrow a rapidly increasing leukocytosis takes place. Then, under influences indefinite and unknown, the upward march is checked, and is followed by a rapid descent. It is possible that the fall in this case



was due to the presence of streptococci found in the blood on September 21st. The organism, of course, may have been present before this date. This was our first bacteriological examination of the blood. The next day the leukocyte count reached its maximum, and the day after that began to drop. At about this time also the temperature began to rise rapidly. It looks as though these two phenomena, rapidly rising temperature and rapidly falling leukocytes, might be due to the invasion by the streptococcus. The leukemic agent, however, is still active, the lymphocytes registering 98.2 per cent. and the polymorphonuclears 1.5 per cent. in a total white count of 6,300. What the differential count at the first examination was I do not know, as, unfortunately, I did not examine any dry cover-slips, the total white count being practically normal. The next examination, however, two weeks later, showed the great increase, 99 per cent., in the

mononuclear elements, which characterized the blood condition up to the time of death, and this condition may have been present on September 1st. It simply shows that in these severe cases of anemia we must not be content with a total white count alone, but must study our dry specimens.

Study of the dried smears prepared with the Wright stain shows interesting blood pictures. The mononuclear cells resemble in structure lymphocytes, having a round or slightly indented nucleus that nearly fills the cells and takes a deep stain. A remarkable number of basophilic granulations are seen. There is great variation in the size of these mononuclears, the diameter ranging from six to thirteen microns, yet the small variety prevails. It is probable that both large and small cells have a common origin, and that the various sizes and shapes are due to violence in manipulation, for the size and appearance of the cells vary in different parts of the same specimen, those in areas where the layer of blood is thick, and where, therefore, they are protected from pressure in the process of spreading, being small and deeply stained; whereas, in areas where the smears are thin, and where, therefore, pressure is greatest, they are larger and less deeply stained, suggesting spherical lymphocytes that have been flattened out. Again, the edges of the larger forms are often uneven and ragged; the cells protoplasm is usually undemonstrable, as if the ectosarc had ruptured, leaving only the round nucleus. That such great changes were brought about by the process of smearing is attributable to the extreme friability of the cells. According to Capps* this friability and consequent cell variation are greater in acute leukemia than in the chronic form.

Red corpuscles show considerable poikilocytosis, increasing with the course of the disease. Nucleated reds are rare.

Following is the report of the autopsy by Dr. Bassoe, pathologist at the Presbyterian Hospital:

Anatomical Diagnosis.—Hyperplasia of intra-abdominal lymph glands, spleen, and intestinal lymph follicles; general anemia; multiple hemorrhages of serous membranes, kidneys, myocardium, and bone-marrow; accessory spleen; agonal intussusceptions of small intestine.

The body is that of a well-nourished child, rather large for his

*Oral communication.

age. The skin is very white. The external lymph glands are not enlarged. The peritoneal cavity is empty and free from adhesions. The pleural cavities are both empty, and there are no adhesions. The pericardial cavity contains a few cm. of clear fluid. The thymus is about 4½ cm. long by 2 cm. wide and 1 cm. thick. The tissue is very pale. Each lateral lobe of the thyroid measures 2½ x 1½ cm.; on section it is pale, grayish-red, and glistening. The mucous membrane of the larynx and trachea is smooth and extremely pale. The tracheal and bronchial glands are moderately enlarged, the largest being 18 x 10 x 3 mm., dark red externally and on section.

The weight of the left lung is 105 grams. The pleura is mottled, light gray and red, with small ecchymoses here and there. It crepitates throughout. The section is very pale and grayish-white. The weight of the right lung is 125 grams, and is in the same condition as the left.

In the heart the epicardium is covered with a large number of ecchymoses. The tricuspid admits three finger-tips, the mitral admits one readily. The valve shows no changes. There are a few ecchymoses in the epicardium of both ventricles and the right auricle. The foramen ovale is closed, the myocardium is light-gray brown generally, but dark-red areas are seen throughout the entire thickness of the ventricular walls. The weight is 100 grams.

The spleen is smooth and weighs 90 grams. It measures 11 x 6 x 3½ cm. On section it is dark red, mottled with whitish bands and dots. At hilum an accessory spleen 1 cm. in diameter. The aorta is smooth. The pharynx was not examined. The esophagus is smooth. The membrane of the stomach is smooth, with numerous reddish areas which do not disappear readily on pressure.

In the small intestine there are six descending agonal intussusceptions. The mucous membrane of the duodenum is pale and bile-stained, with solitary follicles enlarged. Peyer's patches are of a dark-grayish color and prominent, at about the ileocecal valve is a patch 5 cm. long occupying the entire circumference. The appendix is 8 cm. long, and the lining is smooth. The solitary follicles are prominent throughout. In the sigmoid are a few blood-stained crusts.

The liver is 565 grams in weight, and smooth externally. On section it is a light grayish-brown in color, and but little blood exudes on pressure. The lobular markings are invisible, and the

cut surface is of a homogeneous appearance. The gall-bladder contains fluid bile and the lining is smooth. The pancreas is of normal size and extremely pale. The adrenals are small and pale. The kidneys weigh 130 grams. The left measures 8½ x 5 x 3 cm. The capsule is not adherent and leaves a pale-gray surface, on which are a large number of ecchymoses, the largest measuring 6 mm. in diameter. On section it is extremely pale, the pyramids are white entirely, and the cortex markings can be made out in places. The section also has an area of small ecchymoses. There is a narrow reddish line between the pyramids and the cortex. The capsule is thin. The right kidney is the same size and contains a small amount of yellow fat. Otherwise it is the same as the left. The ureters are small. The bladder is extremely pale.

Lymph Glands.—The retroperitoneal glands are moderately enlarged, the largest being 12 mm. in length and very dark. On section it is mottled grayish-red, and in places almost black. Those about the pancreas are the largest, the periaortic glands are of similar appearance. The mesenteric glands are uniformly prominent and dark red in color, but smaller. Cervical, axillary, and inguinal glands are not enlarged, but the iliac glands are slightly so.

The marrow of the sternum is of the usual deep-red color. The marrow of the femur is also very red, with darker hemorrhagic foci.

The brain was not examined.

The meninges of the cord are very pale. The cord is preserved intact.

MICROSCOPIC.—*Heart Muscle.*—The section shows in several places infiltrating hemorrhage into the myocardium. The muscle fibres are forced wide apart by the blood. The number of leukocytes does not seem to exceed much the normal. A large per cent. are of the small mononuclear variety, with deeply staining nuclei. In parts of the section containing no hemorrhage the muscle appears normal.

Right Lung.—In the upper lobe areas are found in which the alveoli are filled with a granular material containing many leukocytes. Between these areas there are areas showing compensatory emphysema. In the first-mentioned areas the walls of the alveoli are somewhat thickened, but not greatly congested. There is a noticeable increase in the nuclei in the walls. The leukocytes within the alveoli vary in kind. There are small mononuclear and

polymorphonuclear and large mononuclear and polynuclear epithelioid cells, some of which contain pigment granules, also some cells which look like plasma cells. The epithelium of the bronchi is not in good condition, but it is in most cases found broken up within the lumina.

Left Lung.—Lower lobe presents similar changes.

Liver.—The liver cells are small and vacuolated, as if they were the seat of fatty degeneration. The spaces between the cords of cells are filled with a granular material. The interlobular vessels are surrounded by connective tissue, which is infiltrated with cells. The intralobular veins appear small. In the one large vein are many cells which have the same appearance as the surrounding liver cells. In one place a small lobule, surrounded by abundant connective tissue, gives the appearance of a cirrhotic liver.

Kidney.—The vessels contain but little blood. The glomeruli show some increase in the number of nuclei, and there are some with ragged outline, and masses of cells with granular material in Bowman's capsule. The capsule is not thickened.

Convoluted Tubules.—The cells are indistinct in outline, and here and there is found a cell within the tubule. The lumen is filled with granular material. The outlines of the straight tubule are in many cases made up of the basement membrane, with epithelial cells scattered within the lumen. Other tubules appear normal. The connective tissue shows no increase, but in many places is packed with leukocytes.

Pancreas.—The pancreas shows very marked necrotic changes, which seem post-mortem in origin.

Spleen.—The spleen presents in the section a mottled appearance, due to areas infiltrated with blood, alternating with areas containing little blood. This makes the Malpighian corpuscles for the most part hard to distinguish. The mottled appearance of the section gives it somewhat the appearance of a section of hemolymph gland. In the hemorrhagic areas it is difficult to tell whether the nucleated areas seen are the pulp cells or leukocytes brought by the blood. The stroma is not to be seen in these areas. There are a few polymorphonuclear leukocytes.

Thyroid.—The thyroid contains a moderate amount of colloid, occurring in masses separated from the walls of the acini by a large space. The epithelium is found desquamated in most of the acini and lying in the space surrounding the colloid. In many cases the acinus is entirely filled with small epithelioid cells, and

there is an extensive cell infiltration throughout. On one edge of the section is a large quantity of pigment, probably the remains of hemorrhage. The suprarenal shows little change. The stomach contains areas of hemorrhage in the superficial layer of the mucosa, otherwise it appears normal.

Small Intestine.—The mucous membrane is necrotic, and throughout nearly the whole length of the section, which is longitudinal, the submucosa is infiltrated with lymphoid cells. Near the muscularis these cells are somewhat far apart. Nearer the lumen they are densely packed, and at the surface they have undergone necrosis, and, with the remains of the mucous membrane, form the lining of the lumen.

Section through a Peyer's Patch.—The mucosa shows the same change as in the other section. The centre of the individual follicles of the patch contain lymphoid cells, widely separated by granular material containing elongated irregular nuclei. The surrounding lymphoid cells are more densely packed, then become more loosely distributed for some distance in the submucosa. Near the lumen they show necrotic changes, as described in the other section.

Thymus.—The organ is large, and the section shows remarkably little change from the infantile type. The corpuscles of Hassal are large, with blue-staining (hematoxylin) centres in many cases. It is lobulated, in the infantile form, but the lobules show no differentiation into cortex and medulla. The lobules are well filled with lymphadenoid cells. Many cells similar to these are found in the connective-tissue septa, and a few in the corpuscles of Hassal.

Accessory Spleen.—The section shows the characteristic splenic capsule, with trabeculæ penetrating the gland. The parenchyma has the same structure as described in connection with the spleen.

Lymph Gland.—The arrangement into lymph cords and channels is not very plainly marked, the lymph cords being so thin and containing relatively few lymphadenoid cells. Around the periphery of the gland the lymphadenoid cells are more abundant. The lymph channels contain many cells of the endothelioid type. These are almost as numerous as the large and small lymphocytes. The number of cells in the channels is not more than normal.

Hemolymph Glands (Retroperitoneal).—Here the well-filled cords of lymph cells stand out in marked contrast to the blood-filled sinuses. The gland is of the splenic type. The circular sinus

is very well made out in most of the periphery. Contrary to the usual finding, the central blood sinuses are packed with blood. This blood contains very many small mononuclear leukocytes, and also large mononuclear leukocytes. A few of what appear to be nucleated red cells are present, but there is no certainty of the identity of these, as there are many lymphocytes containing a minimum amount of cytoplasm surrounded by red cells. No hemolysins could be made out with certainty, but very pale outlines of corpuscles with granular masses surrounding them, the granules staining as hemoglobin, indicate some hemolysis. There is little phagocytosis. In another gland the destruction of red cells is more plainly indicated. The spinal cord was negative; cultures therefrom were sterile.

Bone-marrow.—The bone-marrow from the shaft of the femur and that from the sternum, in contrast to the normal, appear very similar. The marrow of the shaft of the femur shows little of the normal narrow reticulum surrounding fat vacuoles. Most of the vacuoles are absent, and the space is all occupied by a reticulum filled with blood containing large numbers of nucleated red cells (?). There are but few giant cells present. In most places the red cells are disintegrated into a granular material. There appear to be some nucleated red cells present, but, as in the hemolymph glands, these cannot be made out with certainty.

The marrow from the sternum presents a similar appearance, but there are not so many red cells present, and the structure is more plainly made out. There are a few myelocytes present.

Bouillon emulsions were made from the lung, liver, spleen and bone-marrow, and from these glycerin-agar slants were inoculated. No results after seventy-two hours. From the heart's blood the streptococcus was obtained in pure culture. The bile was sterile.

Study of this excellent report of Drs. Bassoe and Dick shows only what has been observed in previous cases. There are noted in organs and tissues generally the infiltration with lymphadenoid cells, closely resembling the small mononuclear lymphocytes seen in the blood during life, showing the widespread distribution of these elements. The involvement of the bone-marrow is confirmatory of the views of Neumann and others, that no case of leukemia thoroughly studied has failed to show an abnormal condition of this structure. Cultures from the bone-marrow, how-

ever, were negative. The presence of streptococci in the blood both during life and post-mortem observed in this case has also been noted in other instances. The enlargement of the thymus noted here has also been observed by Rocaz. It is to be regretted that through an oversight no animal inoculations were made.

Morse's report of 8 cases, McCrae's of 6, and my own of 15 now give us 29 cases of acute leukemia for study. It must be confessed, however, that their analysis adds little to our knowledge of the condition. There seems to be nothing in family or previous history which can be regarded as predisposing to the disease. The family history of tuberculosis in my own case led me to look up this point in others. Unfortunately such history is given in comparatively few instances. In 105 cases 51 make no mention of family history at all, and of the remaining 54 cases 41 are negative, and 14, or 26 per cent., have a family history of tuberculosis. Susmann,[16] studying the relation of leukemia and tuberculosis, even concludes that a certain antagonism exists between the two conditions. The leukemic condition improves, the total leukocyte count diminishing, the neutrophiles increasing. Following this hint, injections of tuberculin have been tried in a few cases, but nothing conclusive has been determined thereby. It is not probable from the data available that tuberculosis plays a part either direct or indirect in the cause of the condition.

As to direct heredity, there have been reported only two cases of the condition in parent and child, too few from which to draw any conclusions.

As with heredity, so with the previous history of cases, nothing appears to act as a predisposing cause, except, possibly, chronic tonsillitis. A large proportion of cases show this condition, but here the question arises whether the tonsillar condition is not the result rather than the cause of the process. It is not improbable, however, that the infectious agents found in the late stages of leukemia gain access to the system *via* the tonsils, and that to this extent at least the diseased condition of the latter may contribute to the final outcome.

No age is exempt. Leukemia has been observed in the newborn child, and as late in life as seventy-one years.

The onset may be sudden, but more often is insidious, with vague indefinite symptoms, which gradually develop into the typical leukemia picture.

The general course of the disease in children, on the whole, is

the same as in adults, and is always fatal. It occurs more frequently in males, 21 of the 29 cases being boys. The spleen was enlarged in all cases, the liver generally, though the enlargement of neither was great. A not infrequent phenomenon was a diminution in the size of the spleen before death. General glandular enlargement was by no means constant, the cervical glands being most frequently involved.

The duration of the cases was from four and a half days to five months. Six, possibly seven, of the cases were prolonged beyond the conventional nine weeks. The onset, however, is usually so insidious that it is impossible to determine exactly when a case does begin. Hence the impossibility of drawing a hard-and-fast line—*e.g.*, at nine weeks as the limit for acute cases. The acute part of the disease is undoubtedly but the final stage of a chronic process, the exact beginning of which we cannot determine.

Nor does the duration depend upon the type of the disease. The most prolonged case, five months, was lymphocytic, whereas the one myelocytic case lasted about two and a half months; and of the two mixed cases one died at the end of four and a half months, the duration of the other was unknown, but was probably short. (Case II.)

As in the adult, the chief interest in leukemia centres in the blood. There is, of course, the diminution in erythrocytes and hemoglobin. The lowest red count recorded is 750,000, and this was after severe hemorrhages. The leukocyte counts ranged from 6,000 to 810,000. Several low counts have been observed, and always just before death. A falling white count, as in my own case, is apparently indicative of approaching death. Age apparently has no effect upon the total white count; thus of two thirteen-months-old-children one had a count of 21,000, and the other 208,000, and the highest count, 810,000, occurred in a twenty-months-old infant.

The differential count showed that of the 29 cases 25 were of the lymphocytic type, 1 myelocytic (Case VII.), 2 of the mixed-cell variety (Cases II. and X.), and 1 case undetermined. The preponderance of the lymphocytic type is thus emphasized anew. The lymphocytosis, furthermore, is more apt to be of the small variety; this cell predominated in 12 cases, the large cell in 3, the two varieties equal in 2, and in the other cases no attempt is made to distinguish between them. The degree to which the lymphocytosis may rise is shown in my own case, where were found con-

stantly 99 and 99½ per cent. of lymphocytes. The degenerated granular character which these cells may undergo is also well illustrated in the same case.

The polymorphonuclears are always reduced, often to an extreme degree. Thus in one specimen from my own case I counted 1,100 cells before finding a neutrophile; in the same specimen also but one eosinophile was seen. The total count here being 80,080, the absolute number of each variety in a cubic millimetre of blood was as follows:

Mononuclears	79,919.8
Polynuclears	80.1
Eosinophiles	80.1
	80,080.0

Whatever the function performed by the neutrophiles, obviously it could not be carried on properly by such a small number of cells.

Except in the myelocytic and mixed cases, myelocytes were rarely present, and then only in small numbers. Nucleated reds were also rare, though found occasionally, especially in young subjects; thus Case IV., thirteen months old, showed eight normoblasts and eight megaloblasts in a count of 200 cells, a striking example of the tendency of the blood in infancy to revert to the fetal type under pathological conditions.

Certain minor differences in the behavior of the disease in early life are observed. Thus, in children hemorrhages are apparently less frequent and less severe; in the 29 cases hemorrhage occurred in only 17, and in only 1 of these was it severe. It occurs in the nose, mouth, bowels, kidneys and skin. The blood picture also represents a greater variety than in adults, and the lymphocytosis is more apt to be of the small type.

In an explanation of the abnormal blood picture, of this turning upside down of the different varieties of leukocytes, lies the solution of the whole leukemic problem. The change is undoubtedly the result of a chemotactic agent acting upon the bone-marrow, the clinical manifestations and type of disease depending upon the nature of this chemotactic agent, whatever it may be. Of the real nature of this agent and of its action upon the bone-marrow we know nothing.

The diagnosis of leukemia can be made only by a complete examination of the blood. Profound anemia and leukocytosis are so frequent and arise from such a variety of causes in infancy and childhood, that there is more chance of error in early life unless one makes a differential white count, the only means by which a diagnosis can be clinched. In the absence of marked symptoms a constant and comparatively high lymphocytic count (90 per cent.) must be observed, owing to the great ease with which a lymphocytosis is set up at this time. Thus I was somewhat disturbed once at finding in a pale twenty-two-months-old infant with hypertrophied tonsils, adenoids, and enlarged cervical glands, a white count of 20,000 and 70 per cent. of lymphocytes. Subsequent examinations, however, showed a normal condition of the blood. Conversely a leukemia with marked throat symptoms may be mistaken for an ordinary acute infection. There is little danger of confusing leukemia with pertussis.

The effect of intercurrent disease or infection upon leukemia is interesting. This subject has been carefully studied by Dock[17] and others, and in a general way it may be said that there is almost always a fall in the total leukocytes, and also, though less uniformly, there is a disposition for the leukemic character of the blood to disappear more or less completely and for the polynuclear to increase absolutely as well as relatively. The total leukocytes have been known to fall as low as 471. Case II. in my own series, for example, shows the effect of a double pneumonia in an eight-months-old boy; we find 28 per cent. of polynuclears and 65 per cent. of total mononuclears. The influence of tuberculosis has already been mentioned. Dock's study shows the effect of influenza, erysipelas, acute and chronic tuberculosis, abscess, etc.

Turning now to the pathological side of the question, the results of autopsies do not throw much light on the problem. Post-mortem examinations in adults and children have shown the widespread distribution of the disease, abnormal appearances having been found in practically all organs and tissues of the body. The condition found most commonly has been an excessive infiltration of tissues with small mononuclear lymphoid cells, identical in appearance with the smaller lymphocytes with which the circulating blood is flooded during life.

Perhaps the most important contributions which have been made to the pathology of the subject are those which have shown that the bone-marrow *only* need be involved in order to produce

the disease, whether of the myeocytic or lymphocytic type. For the researches of Neumann, Walz, Pappenheim, Brandenberg,[18] Kelly,[19] Reed[20] and others have shown that the bone-marrow is *always* involved, the glandular system sometimes not at all, sometimes only slightly, as in my own case; and they maintain that no case which has been thoroughly studied has failed to show a pathological condition of the bone-marrow. The incorrectness of the terms "lymphatic" and "myelogenous" is thus obvious. All cases are "myelogenous"—*i.e.,* of bone-marrow origin. The terms "myelocytic" and "lymphocytic," suggested by Kelly and others, are preferable for the present, even though they are based on a physical sign, *i.e.,* on the presence of either myelocytes or an excess of lymphocytes in the circulating blood.

The description of the bone-marrow given in my own case is typical of the changes found in this tissue. These changes are not always found in all parts of the bone-marrow. They may be localized, and are found most commonly in the shafts of the long bones.

The comparatively few bacteriological investigations which have been made are of a negative value. Streptococci and staphylococci have been found in the blood during life, also these and other familiar organisms have been found in the blood and tissues post-mortem, but no specific organism which can be considered as causing the condition. It is probable, furthermore, that these organisms are secondary invaders. Nichols[21] found in 92 cases showing bacteria that in chronic leukemia organisms were found oftener in the terminal than in the earlier stages. They are probably the result rather than the cause of the original underlying processes, and, as already mentioned, the frequent occurrence of hypertrophied tonsils in leukemic cases suggest that they may have gained access to the system by this route.

Thus, lacking knowledge as to the exact cause of leukemia, various theories have naturally been advanced to explain the disease. Nichols[21] has studied these various theories, grouping them under three heads: (*a*) toxic, (*b*) infectious and (*c*) cancerous or hyperplastic. Of these only the infectious theory has been studied experimentally, and the results here have been negative. Discussing the cancerous theory, he likens the enormous number of leukocytes in leukemia to the proliferation of epithelial and connective-tissue cells seen in carcinoma. He finally concludes that until the true nature of leukemia is understood, both the toxic and can-

cerous theories must be considered among the possible causes of the disease.

Nichols' paper is exhaustive and valuable, but study of it and leukemic literature generally suggests the desirability of further investigations in two directions: injections of blood from living leukemic patients directly into the bone-marrow of animals, and secondly, injections of pure cultures of various pathogenic organisms into the same structure. As far as I have been able to learn from a pretty careful review of the literature, such investigations have not been carried out with pathogenic organisms at all, with leukemic blood but once, and then with a negative result. Accepting as we must the bone-marrow as the primary pathological lesion of the condition, our efforts should be directed toward ascertaining the nature of the agent producing that marrow change. Fascinating and absorbing as is the study of leukemic blood, the condition found there represents, after all, only one physical sign of the disease. The real question is, *why* do the bone-marrow and the other blood-making organs produce this kind of blood, *what* is the agent causing the organs to manufacture 99 per cent. of lymphocytes instead of 40 per cent., or to set free in the circulation 27 per cent. of myelocytes instead of an occasional one?

Treatment is of no avail. Arsenic is generally given, but is useless. The record of cases treated with the x-ray thus far gives no promise of permanent help. The hope that such treatment may be of service is based presumably on the theory that leukemia is a malignant disease of the bone-marrow. Granting the truth of this there is less chance of the improvement seen in more superficial malignant disease, owing to the fact that calcium salts offer greater resistance to the passage of x-rays than do other constituents of the body; hence the greater difficulty of affecting bone-marrow.

The natural behavior of leukemia in patients attacked with an intercurrent disease, suggests a method of treatment not hitherto tried to any extent. If the fall in total leukocytes, the attempt to resume a normal leukocyte formula, and the reduction in splenic enlargement, phenomena already mentioned, in reality be due to the newly invading agent, it seems not unreasonable to hope that injections of sera, anti-streptococcic, anti-pneumococcic, etc., might accomplish the same result, and thus bring about an amelioration of the most prominent pathological conditions of the disease. True, no case thus complicated has ever recovered, nor

has tuberculin been of permanent value; still in a disease as obscure and as uniformly fatal as is leukemia, any methods of treatment are justifiable. Sera injections seem worthy of trial.

CONCLUSIONS.—All cases of leukemia are myelogenous in origin. As in adults, the acute variety is more apt to be of the lymphatic type.

The disease is rare in early as well as in later life. It is probably more common than has been supposed, however, 22 cases of the acute or subacute variety having been reported since 1898, while previous to that date only 7 cases are on record.

The course in children differs from the adult type only in minor details; hemorrhages are fewer and less severe, the lymphocytosis is more apt to be of the small variety. The disease is always fatal.

It is suggested to use injections of sera in treatment.

Experimental inoculations with leukemic blood and with cultures of pathogenic organisms are desirable, such inoculations to be directly into the bone-marrow.

I am much indebted to Professor Hektoen, Drs. Bassoe and Dick for the thorough pathological report, and to Dr. Joseph A. Capps for valuable help in the examination of the blood.

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CEREBROSPINAL MENINGITIS.

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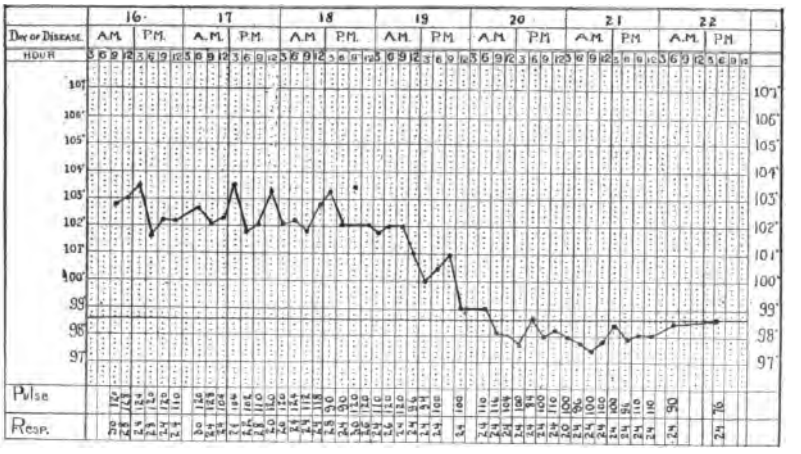
A restrospective consideration of the salient features of cerebrospinal meningitis, based upon a study of 100 cases, observed during the epidemic of 1904, may not be without interest at the present time. No detailed statistical report will be given; the clinical features only are discussed in this article. A study of 60 cases (three-quarters of these were under fifteen years of age) has been presented in the report of Gouverneur Hospital; the others were observed in the Children's Wards of Beth-Israel and Roosevelt Hospitals.

In the first case coming under observation at Beth-Israel Hospital, January 6, 1904, and in but two others, a history of a prodromal period was obtained. There were malaise, headache, loss of appetite, pains in the bones, chilliness and slight rise of temperature.

The invasion in the others was sudden, in many the exact hour was given. Two or more of the following symptoms were noted: Chills, or chilly feelings, more or less fever, convulsive movements or general convulsions, twitchings of a group of muscles or limb, vomiting more or less severe, headache, pain and tenderness of the muscles with rigidity of the back of the neck, irregular pains in the joints or other parts of the body, nasal catarrh or conjunctivitis, restlessness, sleeplessness, active or passive delirium, prostration, peculiar pallor with sunken eyes and greater or less degree of apathy, stupor or even coma.

The initial symptoms do not, as a rule, give us a clue to the subsequent course. We have seen cases beginning with high temperatures, coma and other evidences of intense infection, recover

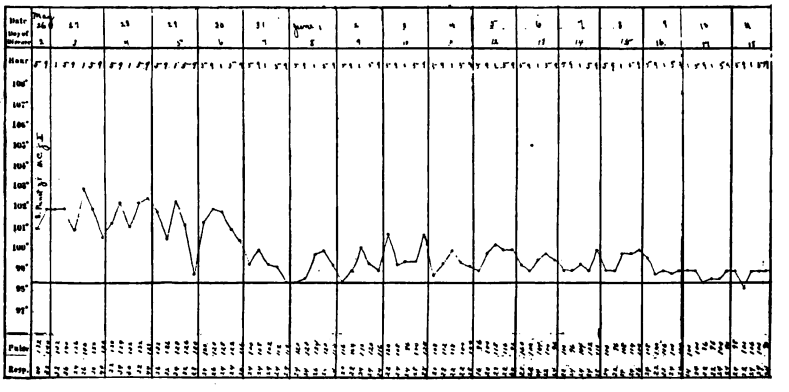


TEMPERATURE CHART I. MILD TYPE.

in a short time and leave the hospital in a fortnight. Such a case was that of which Temperature Chart I. is a record.

History.—Benj. T., nine years old. Onset sudden, with severe headache, vomiting, delirium, restlessness, stupor, etc. Black vomiting on the second day; incontinence of urine and feces. Leukocytosis, 26,000. On the third day 15 cc. of fluid escaped on lumbar puncture, showing positive diplococcus. Herpes on the fifth day. On the day following lumbar puncture there was decided improvement in the general symptoms. The case ended in complete recovery, although the onset was severe.

Another example of the mild type of the disease is shown in the Temperature Chart II.



TEMPERATURE CHART II. MILD TYPE.

Temperature Chart II.—Philip M., three and one-half years old. Onset with headache, vomiting and restlessness. *Condition on admission* (thirty hours from the onset), unconscious, quiet; slight pallor. Pupils small and inactive; neck rigid; Kernig's sign present. Temperature 101° F.; pulse, 132; respiration, 40. Numerous petechias on body. *Complications:* Hematoma on dorsum of right hand, appeared on the third day, and resolved in about two weeks. Abscess in lumbar region following hypodermoclysis, ten ounces of saline being injected on the second day. Lumbar puncture on day of admission gave one dram of turbid fluid; diplococci present.

Remarks.—The patient became conscious and irritable on the third day; on the fifth day there were twitchings of the left side of the face and the left arm. On the sixth day the pupils were normal and there was but slight rigidity of the neck, though Kernig's sign was marked, and, in fact, persisted for about three weeks. There was steady improvement and the temperature remained low after the sixth day. The pulse was from 100 to 150, up to the sixth day; after that, 90 to 124. On the nineteenth day the temperature fell to 97° F.; from then on, until the thirty-fourth day, it was practically normal.

On the other hand, a comparatively mild onset was often followed by a more or less prolonged convalescence or a fatal termination. In the words of J. Lewis Smith, "there is probably no disease which falsifies the prediction of the physician more frequently than cerebrospinal fever."

Chilly feelings were common early symptoms in children three to four years of age and older. Later on in the disease irregular chills and chilly sensations occurred at irregular intervals in many cases. They were of no prognostic import, for some of these patients eventually recovered.

Pain in the head (mostly frontal, at other times occipital, parietal or general) was a constant and distressing symptom. Even when unconscious, from time to time, there would be a cry: "Oh! my head." Pain, tenderness or tenderness and retraction of the posterior cervical muscles (in mild cases a resistance to the forward movement without interfering with rotations) was an early and pathognomonic sign.

In severe cases the vertebral muscles were involved, causing orthotonus more commonly than opisthotonus.

"The usual position of the patient in bed, in a typical or

marked case, is with the head thrown back, the thighs and legs flexed, with or without forward arching of the spine. The muscular retraction and rigidity continue from three to five weeks, more or less, and abate gradually; occasionally they continue much longer."—(J. Lewis Smith.)

In several of the Gouverneur Hospital patients the condition persisted for three to four months. In 1 case (still under treatment with temperature) more than five months have elapsed, from the time of the first appearance of contractions. Two chronic cases of over five months' duration still remain in Beth-Israel. The symptoms, without doubt, are due to the presence of a serous effusion in the ventricles and subarachnoid space.

FIGURE 1. (DORA C.)

An excellent example is presented in the case of Dora C., one year old. (Figure 1.)

Any attempt to move the contracted muscles induced severe pain. To develop Kernig's sign (present some time or other in nearly all cases) was painful and increased the restlessness in many instances. Pain along the spinal nerves, changing from one part to another, was frequently observed, as was hyperesthesia of the skin. In 4 of the Beth-Israel cases the abdominal pain was very severe and gave rise to a good deal of complaint. Later on insensibility and numbness followed the hyperesthesia and painful stage.

General convulsions were common in children at the onset, and later on in severe or fatal cases. Localized (convulsive) seizures were noticed about the face in particular; less often one extremity or one-half of the body was involved. In several there was difficulty in deglutition, which, however, was of short duration, disappearing within thirty-six to forty-eight hours, and probably due to muscular paresis. Strabismus, ptosis and loss of power of the sphincters of the bladder and rectum with incontinence of urine and feces, were frequent. Monoplegia and hemiplegia have been observed.

Conjunctivitis and otitis (catarrhal or suppurative) were common. The process is probably due to the extension of the infection from the nasal mucous membrane, as the characteristic diplococcus has been detected in the secretion from the eye, nose and ear. Furthermore, serious affection of the eyes, of the internal structures of the ear, with loss or impairment of sight or hearing, may be occasioned by central causes in the brain or special nerves.

Photophobia and contracted pupils, present in the beginning, were followed by dilatation and absence of pupil reflex in the later stages, with the increase in the intracranial pressure. Inequality

FIGURE II. (WILLIE G.)—EMACIATION, WITH RETRACTION OF ABDOMEN. TEMPERATURE CHART V. ON PAGE 100 REFERS TO THIS CASE.

of the pupils, hippus, feeble response to light were quite frequent. Nystagmus was fairly common.

Hearing, very acute in many cases at the outset, became dull, or was, perhaps, entirely lost later on.

Prostration, with general relaxation, was an early evidence of intense infection. In the later stages it was due to exhaustion, or the result of trophic disturbances. Emaciation, with retraction of the abdomen, was a common symptom in chronic hydrocephalus following cerebrospinal meningitis. (Figure II.)

Restlessness, sleeplessness, delirium, apathy, tremors, stupor and coma occurred early or late, or were present to a greater or less extent throughout the progress of the disease.

The nervous manifestations were mild or pronounced and violent, necessitating restraint at times.

The mental condition varied in individual instances. In some

we noticed slight drowsiness, a sleepy state, apathy, varying degrees of stupor and finally profound coma.

Delirium may be mild or violent and associated with extreme restlessness and irritability. Now and then the sensorium is involved only for a short period. Coma may persist for a long time, it may be followed by recovery or there may be more or less improvement succeeded by a relapse; these phases may be repeated time and again. Finally, death takes place.

Many of the older children, as soon as the acute stage with its pain and hyperesthesia had passed, appeared to be quite contented in their "coiled up" position: when asked, "How do you feel?" they would answer, "I am better" or "I feel well"; although the temperature and muscular rigidity belied the words at the time.

Vomiting, as an initial symptom, was rarely absent. Later on, in rare cases, it was present as an evidence of nephritis; more commonly it was due to the inflammatory processes at the base of the brain or was symptomatic of chronic hydrocephalus. At times it was a very distressing and dangerous symptom. When the muscles of deglutition were involved, there was inability to swallow, with regurgitation of whatever was taken. In some cases of the fulminant type, blood was vomited. In the case of Benny I., recovery occurred, though he had vomited quite an amount of grumous fluid.

The appetite is generally poor, the tongue is more or less coated, now and then covered with a thick white fur. At times sordes covered the teeth and tongue, not infrequently the organ was bright red or glazed.

The bowels are generally constipated. In the prolonged cases, particularly with chronic hydrocephalus, more or less incontinence was present.

Though it is claimed that the spleen is enlarged in the acute stage, it was difficult to verify the statement. The general hyperesthesia, complaints of abdominal pains and tenderness with an increase in the general restlessness and irritability, caused by any attempt at careful palpation of the abdomen, made it impossible to examine with the necessary care.

Breathing was but moderately accelerated. Cheyne-Stokes and sighing respiration were present at times. Pulmonary edema or pneumonia as a complication gave rise to characteristic rapid and labored action. Evidences of respiratory failure from central

causes were observed now and then, and were the direct cause of a fatal issue.

The pulse was slow, as a rule, in adults; in children, on the contrary, it was extremely rapid. The characteristics observed in tuberculous meningitis were not found in the acute forms of cerebrospinal meningitis.

There is nothing pathognomonic about the fever. As shown on the accompanying charts, the temperature ranges and varies within wide limits. A case running a low grade is often as dangerous as one with a considerable elevation. The temperature may be irregular; remissions and recrudescences were of frequent occurrence. No prognostic value can be attached to sudden drops. Subnormal temperatures were frequent in chronic cases.* Erotic symptoms were of common occurrence.

Skin eruptions were observed in a fairly large number of patients. A more or less diffuse mottling appeared upon the face, and other parts. It was noticed particularly about the ears, forearm, hand and lower extremities. At the outset the skin was of a peculiar ashy pallor, with dark circles about the eyes.

In restless patients, the integument on the exposed parts, elbows, trochanters, etc., was covered with minute fine punctations, more or less diffuse, resembling superficial abrasions. In others, parallel hemorrhagic streaks due to scratching with the finger nails were found.

A "tâche cérébrale" was readily evoked in the majority of instances. Irregular areas of congestion due to vasomotor disturbances, occurred upon the forehead, face and other parts of the body.

Petechiæ occurred early and were noted in about one-third of the patients. Ecchymotic spots and purpuric plaques were seen in the severe type and fulminant cases. Now and then petechiæ were observed upon the conjunctivæ, in other cases larger areas of congestion were present independent of, and not associated with, conjunctivitis. In a few chronic cases successive crops of petechiæ were found later.

"Cutis anserina" was not uncommon.

Herpes upon the lips, alæ nasi, cheeks, auricular and post-auricular region occurred frequently about the third or fourth

* See Charts Nos. III. and VI. for decided drops in temperature. In Chart No. V. there was from the thirty-fifth to the fifty-seventh day continuous sub-normal temperature.

day. In one patient, a boy of eight years, in addition to the crop on the lips, a large group appeared over the sternum (as shown in Figure III.). In 2 cases, herpes were present on the fingers.

Roseola, urticaria and blebs over the tip of one or more fingers, filled with bloody serum, were seen now and then. Drug eruptions, due to bromids, phenacetin or other coal-tar products occurred in isolated instances.

Cracked condition of the skin about the joints, bed-sores, branny desquamation, similar to that which occurs in chronic disturbances of nutrition in general and desquamation of the hard

FIGURE III.—HERPES OF STERNUM, LIPS AND ALA NASI.

epidermis of the soles and palms in large flakes, were met occasionally in the protracted cases. Picking at the lips and nares, giving rise to bleeding and ulcerations, was an annoying feature in many cases. A chronic case at the Beth-Israel Hospital, presented an abnormal growth of hair over the extremities and body (Figure IV.) during the two and a half months preceding discharge from hospital. The disease lasted six months and resulted in chronic hydrocephalus with persistence of the "curled-up" position.

Blood examinations were made in nearly all cases. Leukocytosis, principally of the polymorphonuclear cells, varying from 18,000 to 40,000, was found. The condition persisted even in the protracted cases. In 3 cases numerous lymphocytes were detected.

The characteristic diplococcus was found in nearly all cases.

We were able to detect the germ at a very early period, in the cerebrospinal fluid. In one, within eight hours from the onset; in others, within ten or twelve hours. The microorganisms were readily found in the smears in the beginning of the disease, in the chronic cases sometimes only after cultures were made. Thus they have been found as late as the 104th day.

It is claimed that the disease is more common among the poor and in crowded quarters; all sections may be invaded, however.

Our hospital cases were brought in from the densely populated tenement districts. The past winter was exceptionally severe; pneumonia was unusually frequent with a high mortality; times were hard and the rooms were tightly closed. The air was impure in the close apartments, the sanitary and hygienic measures sadly

FIGURE IV. (JAKE)—CHRONIC CEREBROSPINAL MENINGITIS; ABNORMAL GROWTH OF HAIR OVER BODY AND EXTREMITIES.

neglected. With the advent of warmer weather, the epidemic appeared to lessen.

The better sections did not escape altogether, a number of cases were seen in consultation under favorable surroundings.

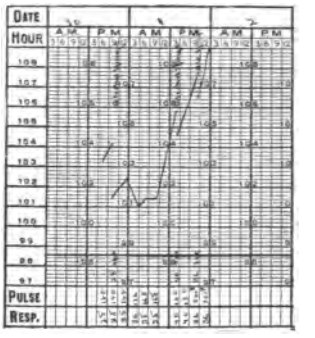
It is generally stated that the specific poison is not transmitted by contact from person to person. It does not seem to spread from one case to another. It usually attacks a number of people at different places, and it is not possible to trace any connection between successive cases. But rarely do we find more than one affected in the same house or family. To this, however, there are numerous exceptions, at least during this present epidemic. More than a dozen instances have come under personal observation, and others have been cited by colleagues in which two were attacked in the same family. In several others, three were taken down in a household, not always at the same time, however.

Our records show that multiple cases were occasionally met with in the same house.

In hospital practice, 2 cases developed a number of weeks after they had been admitted to the ward for other troubles. In both, the diplococcus intracellularis was found in the spinal fluid. One child, a girl, was four years of age, the other a little boy but eight months old.

The *fulminant type* begins with profound toxic symptoms (usually with eruptions), and runs a rapid fatal course in from five hours to several days. The temperature may be moderately elevated or high. In one of our patients it reached 109° F., as shown in the accompanying Temperature Chart No. III.

Temperature Chart III. Malignant Form.—Thos. G., three and one-half years old. Admitted eight hours after onset. Death in forty hours. *Temperature 109° F.*



TEMPERATURE CHART III.

The necropsy showed marked congestion of vessels; diffuse general cloudiness of pia (most marked over cortex); considerable serous exudate present.

In the *ambulant* and the *aborted* cases, though the initial manifestations may be stormy, in the latter the symptoms disappeared within a few days and recovery was prompt. Now and then, within a few days or a week, particularly if the patients were imprudent, a relapse would take place, the case running a more or less prolonged course, with the usual incidental dangers.

In the *severer infections* the temperature (more or less irregular and variable) persists for eight or ten days or even two weeks; then the general symptoms begin to mend in favorable instances or convalescence may be delayed, by various complications and sequelæ.

Osler claims "that a sudden fall in the temperature is of bad omen." Several of our temperature charts, presented herewith at the end of the article, show decided drops and still recovery took place. In one case there was a drop of 9.2° F. (See Temperature Charts Nos. IV. and VI. at the end of this article.)

Complications such as nasal catarrh, conjunctivitis and otitis were frequent; bronchitis and pneumonia occurred now and then. Pleurisy, pericarditis or endocarditis and joint affections were

not encountered in our series. Chronic hydrocephalus, blindness, loss of hearing or speech, various degrees of mental impairment and different forms of paralysis were met as sequelæ. Cervical adenitis without suppuration was seen a number of times at the Beth-Israel Hospital. In only one instance, in an adult, parotitis was present.

Cases of chronic hydrocephalus were quite frequent and were characterized by periods of improvement, followed by a return of the unfavorable symptoms. Progressive emaciation, sometimes quite rapid, irregular temperature, alternating coma and delirium, incontinence of urine and feces during the relapse, spells of vomiting, dilated pupils, marked retraction of the abdomen (as shown in Figure II., Willie G.), are the prominent symptoms. In addition, headache (paroxysmal in character) and convulsions at times were observed in several cases at Gouverneur Hospital.

The youngest child in our hospital service was about six months of age; in private several were seen thirteen, fourteen and fifteen weeks old. Recovery was observed even at this early age. Chronic hydrocephalus was very apt to follow, as a sequel.

Jacob J., eight months old, had been ill for three and one-half months. The onset was sudden, the subsequent course irregular, but progressive, at times some improvement, then relapses. Admitted to the hospital on March 23d, he died the next day. (Figure V.)

		P.	R.	T.
March 23d—12.45	P.M.	?	48	95°
3	P.M.	88	48	94°
6	P.M.	112	48	95.6°
9	P.M.	104	32	95.8°
March 24th—12	P.M.	100	44	104.8°
3	A.M.	152	36	102°
6	A.M.	148	36	100°
9	A.M.	112	32	97.4°

The photograph shows the emaciation, large size head, and marked degree of retraction, etc. Shortly before death the occiput and sacrum were in apposition. No autopsy. Postmortem puncture allowed escape of over one-half pint of cerebrospinal fluid, in which the meningococcus was still found.

Success attending the treatment is limited, for the actual cause has not been discovered. The source of infections, and the

method of transmission are unknown, nor are we familiar with the manner in which the organisms are eliminated from the body. We are not in possession of any data which enable us to resort to

FIGURE V. (JACOB J.)—CHRONIC HYDROCEPHALUS, ADVANCED STAGE, EXTREME OPISTHOTONUS.

preventive measures, to adopt proper prophylactic measures. When we consider the nature of the lesions, presented by the disease, and bear in mind the intensity of the initial infection, we must conclude that the object of the physician is prevention rather than cure. It is true, the diplococcus intracellularis meningitidis

has been discovered. As the organism is frequently detected in the nasal mucous membrane, it is inferred that the infection enters through the nasal lymph channels, and by them is conveyed to the meninges and subdural space. The possibility of the blood carrying the microorganisms must not be lost sight of.

Blood cultures made by competent bacteriologists have revealed the germ in the circulating fluid. Little is known of the life history of the diplococcus, outside of the body, and many interesting problems are still unsolved. It is to be hoped that the mysteries of the mode of infection will be elucidated. This much dreaded disease of childhood, and early adult life, cannot be blocked at the fountain head, until the causes and the manner in which it becomes diffused among the well in an epidemic form are discovered.

In the discussion of the value of any plan of treatment of cerebrospinal meningitis of the epidemic variety, several cardinal points must not be lost sight of, viz.: (*a*) the great irregularity in the clinical course of the infection; (*b*) the high rate of mortality in most epidemics.

The malignant cases (fulminant or apoplectic type) die within twenty-four to seventy-two hours, some after five to twenty hours' illness. In such, our therapeutic measures are of little avail. In Gouverneur Hospital, one of our patients died within fifteen hours. In addition to the ordinary form there are anomalous types. The attack may be ushered in with severe symptoms; in a day or two they subside and convalescence is rapid. Then, again, there are certain mild cases, in which headache, nausea, and more or less cervical pains, stiffness or rigidity are noted. The fever is moderate or absent, and a diagnosis is only possible during the existence of an epidemic.

In the intermittent form, we have exacerbations of fever, recurring every day or every other day. The curve being of an intermittent or remittent character.

In the chronic variety the attack may be protracted for several months. There is intense marasmus, a series of recurrences of the fever, and a complex symptomatology attributed to abscess of the brain, or chronic hydrocephalus.

The clinical course varies, as we have observed, within wide limits from a few hours to four months or over. There is a case in Gouverneur Hospital with irregular temperature after 150 days' illness. Osler writes: "The high rate of mortality which has ex-

isted in most epidemics indicates the futility of the various therapeutic agents which have been recommended." The present epidemic is no exception. The mortality was high, the percentage of malignant cases large. Strong robust children were attacked more frequently than were the puny and weak. The greatest mortality was in children under five years of age. The larger proportion died within the first five days. Several cases in infants twelve, fourteen and fifteen weeks old, seen in consultation, recovered from the meningitis, though chronic hydrocephalus followed as a sequel.

Exacerbations and remissions were frequently observed after apparent convalescence; unexpectedly a recrudescence or a relapse would follow a short period of improvement in the general symptoms. Unfortunately, these mishaps could not be foreseen or guarded against.

Each case must be judged by itself. The irregular and variable course of the disease in different patients leaves us in the dark as to the relative success of treatment. It is not in our power to destroy the germ, nor can we at present counteract its toxic effects.

The strength of our patients must be kept up by proper nourishment and careful, skilled nursing. This is of the utmost importance, particularly in the protracted cases.

The symptoms in general must be treated as they arise. Whenever possible the patient should be isolated. Rest and quiet, for both the mind and body, are essential.

Potassium or sodium iodid given from the inception of the attack, or perhaps not until a later stage, has been advocated by various authorities. In the *Medical Review of Reviews* (May 25, 1904) the use of the iodids is insisted upon. The assertion is made that under their continued administration all the products of inflammation finally disappear, the mortality being small. In one of our large hospitals, the plan was given a thorough trial. The remedy was used in increasing doses over long periods. Blindness and deafness persisted in some of the cases that recovered. The fatality was about the same. Ergot has its ardent supporters. Morphia, codeia or opium is of material benefit in controlling the pains, restlessness, etc. In severe cases morphia hypodermically is absolutely necessary. Bromids have been employed for a similar purpose. Hot packs or baths (at 90°, 95° or 98°), with or without mustard, relieve the irritability and restless-

ness and frequently promote a quiet sleep. The muscular spasms, retraction of the head and rigidity in general are greatly lessened by the bath. Leeches had been applied before admission in many without material benefit.

Phenacetin in small doses, repeated at intervals relieves headache and pain. Ice-bags to the head and in some cases to the spine, were resorted to as a routine measure. Ointments (Credé, mercurial, etc.) were employed without any appreciable effect.

It may be well before entering upon a discussion of lumbar puncture to refer incidentally to the lesions met with in fatal cases. We may have an acute inflammation of the meninges with more or less involvement of the superficial layers of the brain and cord.

The pia is infiltrated, serum, fibrin and pus are present in varying proportions and quantities in different instances. In children, in particular, the ventricles contain a smaller or larger amount of fluid.

The cortex may be edematous, softened or infiltrated with pus cells.

In some, the pia is congested, or there is an increase in cells, without any exudation.

The inflammation may involve the cranial and spinal nerves, the most marked lesions being in the second, fifth and eighth nerves.—(Delafield.)

In a personal communication, Dr. O. Schultze, who, as coroner's physician, has made a number of autopsies in the rapidly fatal cases, says: "In these cases I am impressed by the evident amount of compression of the cortex, apparently due to increase of fluid in the ventricles. It seemed to me that this compression was the direct cause of death in these cases, rather than the toxemia from the infection; basing my opinion upon the short duration of the disease, temperature and moderate or slight development of degenerations in such organs as heart muscles, kidneys and liver."

To recapitulate, in a certain proportion (*a*) we find an intense congestion of the pia and blood vessels, without any exudation. In others (*b*) a variable amount of serum, pus or fibrin is found. In children, in particular, the lateral ventricles are distended, more or less, and an excess of fluid is present in the subdural space.

Aside from any fault in the technique of lumbar puncture, a

dry tap will result if the former condition (*a*) obtain. In the latter (*b*), by means of this simple procedure, we are enabled to gain a definite knowledge of the character of the inflammatory products; whether serous, serofibrinous, seropurulent or purulent. Sometimes a plug of fibrin occludes the needle or the communications between the lateral ventricles and the subarachnoid space may be shut off by adhesive inflammation. In such circumstances, the puncture is negative.

In serous meningitis, lumbar puncture is of the greatest possible benefit, as shown in the reports of cases cured. When fibrin and pus are present, plus toxic symptoms, the problem is more complicated. It is no longer a simple question of the removal of fluid. We are dealing with a toxemia in addition to the exudation of fibrin, pus, etc.

More or less serum may be readily removed; fibrin, pus and other products of inflammation remain. The toxic symptoms are not materially influenced by the procedure.

The amount drawn off varies from a few drops, enough for a microscopic examination, to 8, 10 or 14 drams. The fluid may be clear, more or less turbid, yellowish, greenish, or distinctly purulent. At times it escapes drop by drop, or more or less rapidly in jets or a stream. Only when it was thought that the needle was occluded by a plug of fibrin did we venture to resort to gentle aspiration with a hypodermic syringe.

Lumbar puncture was resorted to in quite a number of cases. The results have been disappointing from a therapeutic point of view. As the result of a wide and varied experience, the writer is forced to take a conservative view and endorses fully the position taken by Jacobi.

"Lumbar puncture, mostly between the third and fourth lumbar vertebræ, has resulted in facilitating a diagnosis in many instances, and in temporarily relieving some symptoms; those of congestion, edema, pressure, coma, but rarely aided in accomplishing a cure."—(*Therapeutics of Infancy and Childhood.* Page 278.)

The brilliant results reported to have followed the use of lysol injections have not been realized in our hospital cases, in which this method has been tried. After using the plan a few times, with indifferent results, it was discarded.

In a series of early cases, presented in detail in the Gouverneur Hospital Report, the results obtained by *early lumbar puncture*

and *hypodermocylsis* are given. Normal salt solution at the temperature of the body was injected in the hope of diluting the toxins and possibly favoring their elimination. The results were rather encouraging.

The following case is interesting, inasmuch as it was one in which permanent drainage was accidentally established:

Michael R., four years old (brother died of meningitis on May 10, 1904) was admitted into the hospital six hours after initial symptoms of vomiting and headache.

He was in coma, neck rigid, tâche marked, temperature 102.4° F. Lumbar puncture at 8 P.M. (twelve hours from onset), 5 c.c. turbid fluid removed, containing numerous diplococci. From this time on (May 10th) the puncture drained more or less continuously and slowly (drop by drop). Nineteen days later the puncture had apparently closed, and an area of redness and swelling appeared over the site.

May 30th, a slight incision permitted the escape of considerable turbid fluid. A sanious discharge for twenty-four hours followed, upon making a larger incision the next day. Under the application of wet dressings, the inflammatory symptoms referred to above quickly disappeared. From this time on until death, June 18th, cerebrospinal fluid containing diplococci continued to discharge.

From May 29th, basal symptoms appeared, the child was unconscious, emaciated rapidly; obstinate vomiting set in and death occurred on the fortieth day. Practically the drainage was continuous, interrupted by occasional closure of the opening for brief intervals.

Throughout the course of the disease, the flow was seropurulent in character, containing numerous polymorphonuclear cells with diplococci. At no time were there any evidences of a mixed or accidental infection of secondary origin.

The suggestions to trephine the skull, do a lumbar puncture and wash out with a normal salt solution could not be adopted, as we could not obtain the consent of the relatives, in what we deemed were appropriate cases.

In this connection the writer takes pleasure in presenting briefly a *résumé* of some cryoscopic examinations of cerebrospinal fluid removed by lumbar puncture made by Dr. J. H. Bailey (Senior house physician, Beth-Israel Hospital).

Normally, the point of coagulation of serum is about —.56° C.

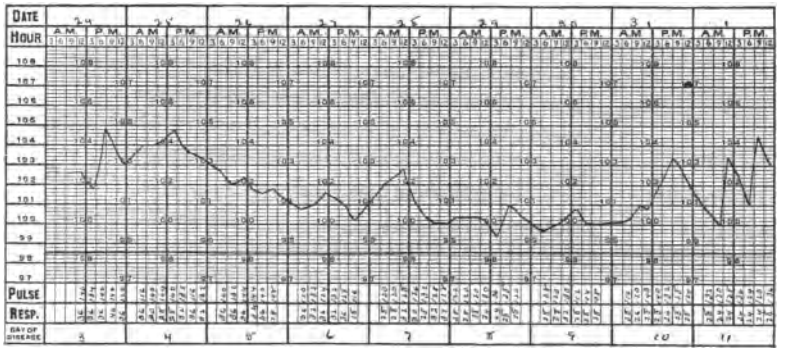
That of cerebrospinal fluid is lower; it varies between —.72 and —.78. In diseased conditions it varies between —.56 and —.74, and in a large majority of cases between —0.60 and —0.65.—(Chavasse and Maher, *American Journal of the Medical Sciences,* May, 1904, page 925.)

The following is a brief report of Dr. Bailey's studies:

The limits of the freezing point (in a study of 69 cases) were —.815 and —.500. The vast majority of cases, however, 79 per cent., ranged from —.64 to —.52, a variation of only —.12. The average point was —.575. The greater part of the depression of average freezing point was —.575. The greater part of the depression of the freezing point is due to the sodium chlorid content. The freezing point varies not only in specimens from different cases, but, also, in specimens from the same case at different times. The depression of the freezing point gives no information as to the prognosis.

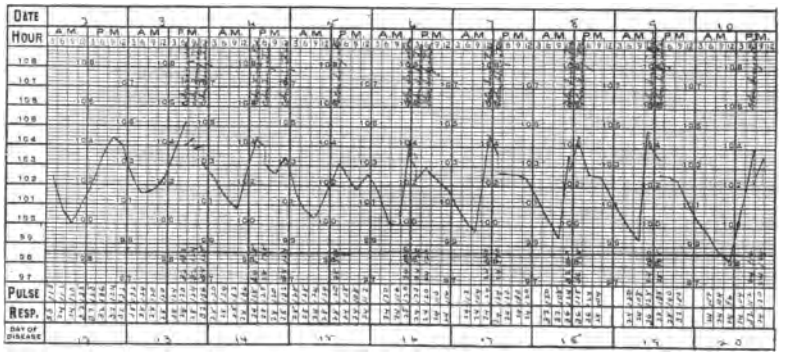
The following temperature charts illustrate certain phases of the disease. Charts IV. and VI. show the irregular type of temperature with decided drops and sub-normal temperature, followed by recovery. Chart V. shows the course of chronic cerebrospinal meningitis followed by chronic hydrocephalus.

Temperature Chart IV. Irregular Type.—Max M., two and one-half years old. Onset sudden (fever, extreme restlessness, delirium, severe pain in head and neck). Petechia. Noisy when admitted, extremely restless, comatose. Noisy for four days, then became quiet—consciousness returned gradually. Somewhat apathetic for weeks, and very irritable when spoken to. From tenth day on temperature intermittent in type. From twenty-fifth to twenty-seventh, subnormal, at times 96.95°; twenty-eighth and twenty-ninth, recrudescence, then low and subnormal, followed by exacerbation on thirty-third day (vomiting, increased headache, moderate coma), lumbar puncture followed by decided improvement next day. Subsequent course favorable. Remission from latter part of seventh day to latter part of tenth day. Pus from abscess of thumb showed some diplococci: no diplococci intracellularis found. Drop in temperature. Remission thirtieth to thirty-third day. From June 29 to July 6, the temperature was normal.



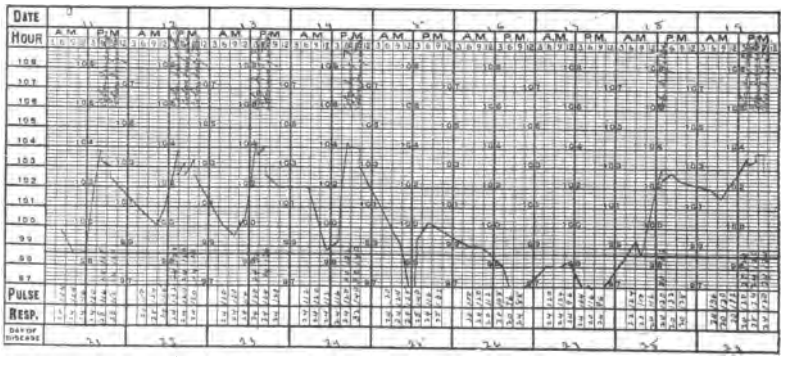
TEMPERATURE CHART IV., 1.

Some diplococci, not intra-cellularis, found in pus from abscess of thumb.



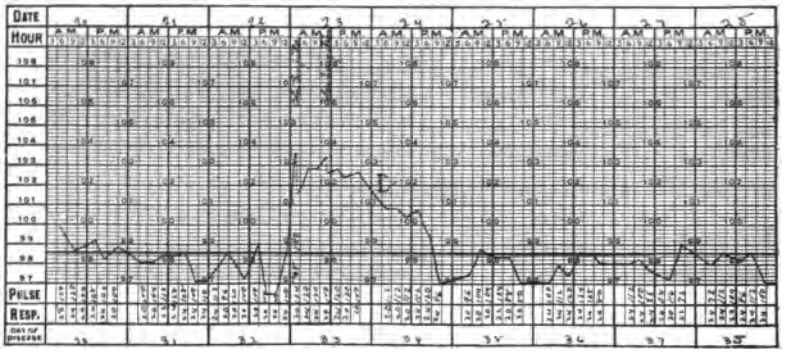
TEMPERATURE CHART IV., 2.

Lumbar puncture drew off fluid free from cells.



TEMPERATURE CHART IV., 3.

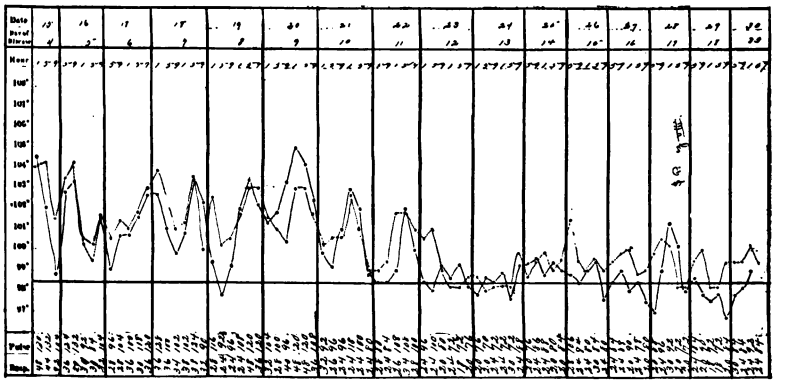
On the twenty-fourth day the 6 P.M temperature was 104.2° F., on the twenty-fifth day at noon, it was 96° F., a fall of 8.2° F. There was then a remission of the fever until the twenty-eighth day.



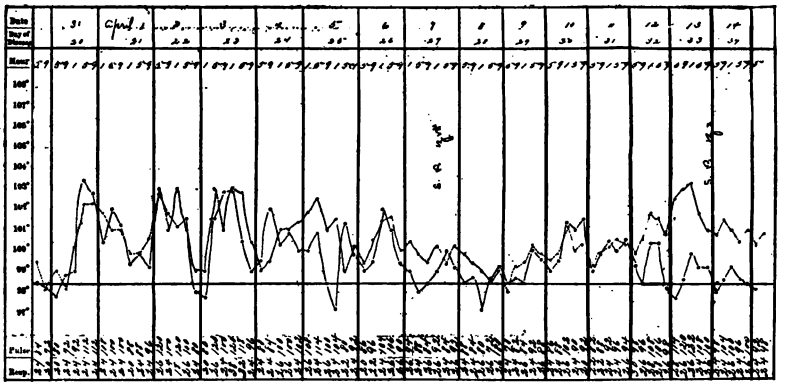
TEMPERATURE CHART IV., 4.

Remission from the thirtieth to the thirty-third day. On the thirty-fourth day the spinal fluid showed meningococci.

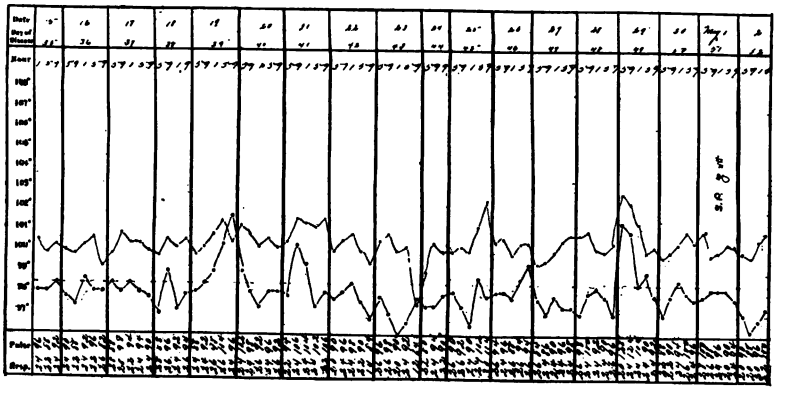
Temperature Chart V.—Willie G., six years old. Prolonged course ending in chronic hydrocephalus, with death on the one hundred and nineteenth day. The marked emaciation and retraction of the abdomen are shown in Figure II. (page 85), which is from a photograph of this patient.



TEMPERATURE CHART V., I.



TEMPERATURE CHART V., 2.



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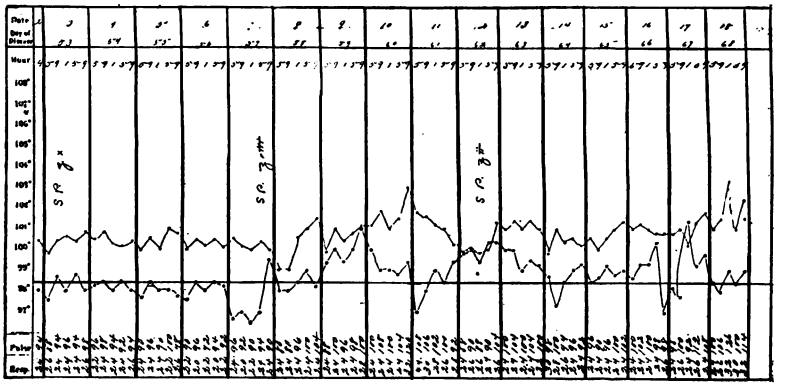

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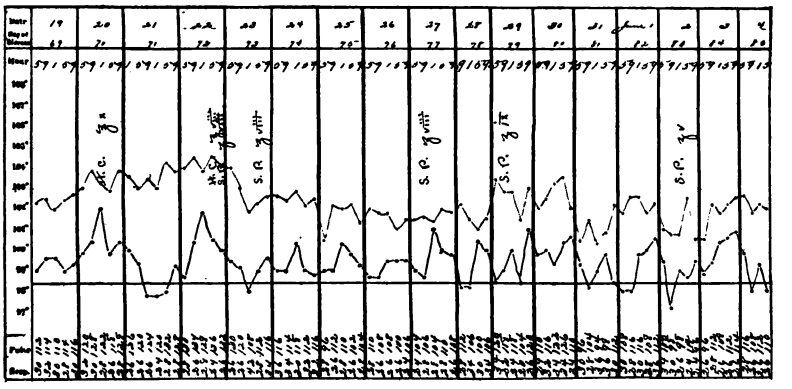

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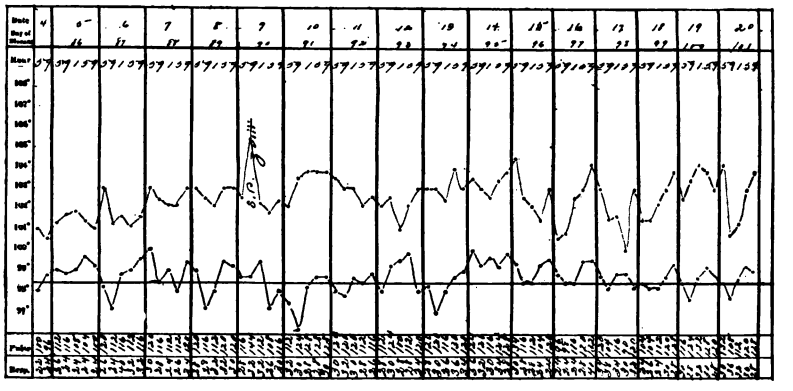

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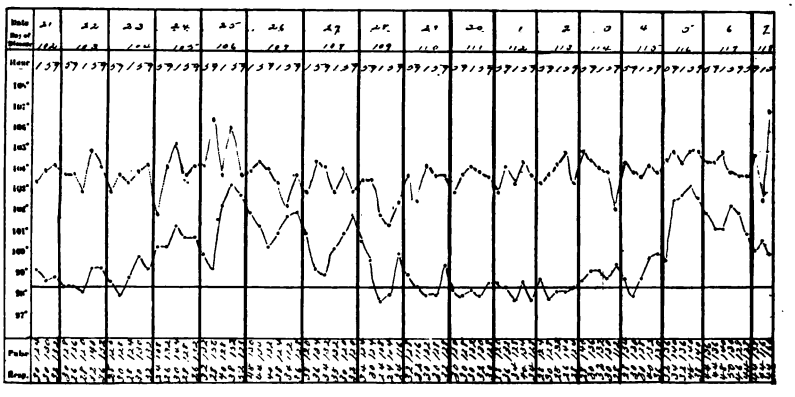
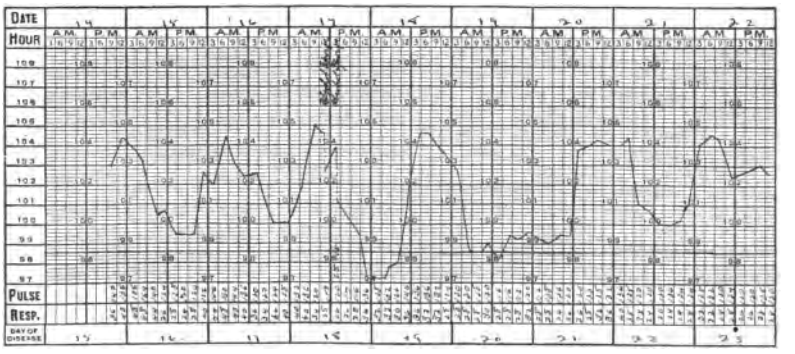
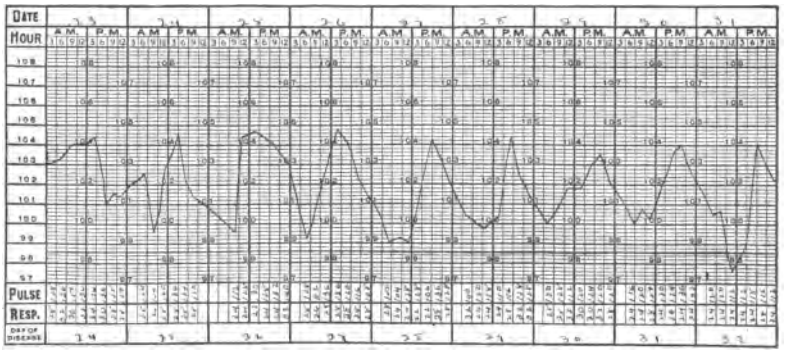


TEMPERATURE CHART V., 7.

Temperature Chart VI.—Shows an irregular type of temperature, with recovery. The patient, Rachel A., was three and one-half years old: the onset was acute and the child was admitted to the Roosevelt Hospital on the fourteenth day of the disease. There was double metastatic choroiditis on the seventeenth day, with resulting loss of vision.



TEMPERATURE CHART VI., 1.



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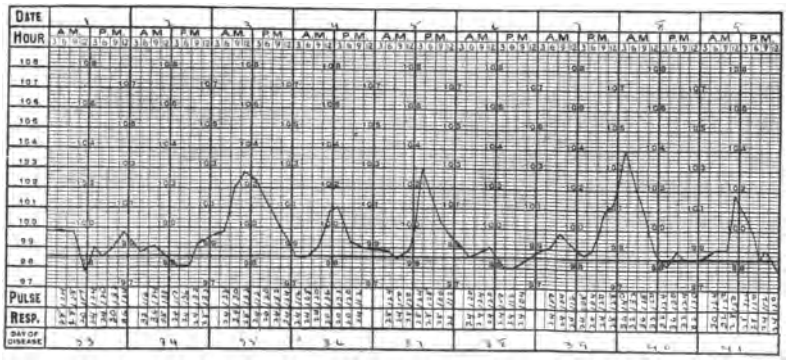

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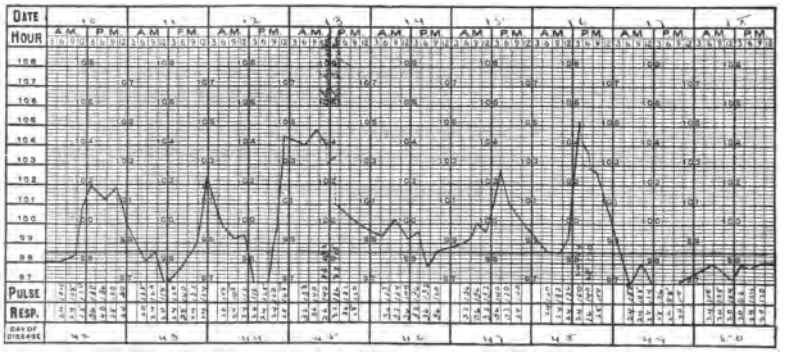
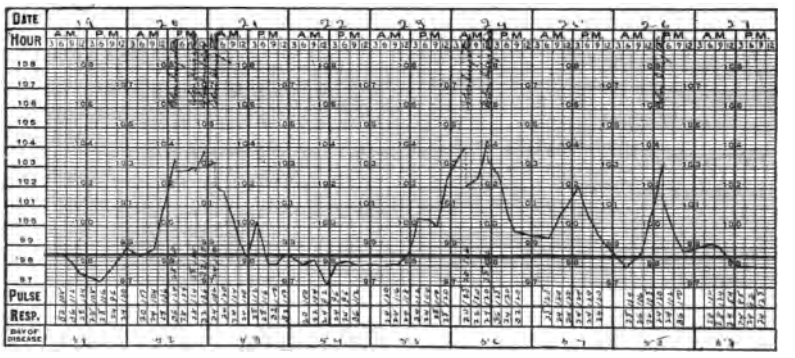


TEMPERATURE CHART VI., 4.

On the forty-fourth day, at 6 P.M., the temperature was 96° F.; at noon on the forty-fifth day it was 104.4°F., a rise of 8.4°F. On the forty-eighth day the 3 P.M. temperature was 105.2° F., on the forty-ninth day at noon it was 95.8°F., a fall of 9.4° F. in less than twenty-four hours.



TEMPERATURE CHART VI., 5.

There was a remission on the forty-ninth, fiftieth and fifty-first days, a recrudescence on the fifty-second and fifty-third and fifty-fifth to fifty-eighth days, followed by convalescence. The temperature was practically normal after the sixtieth day.

PERCUSSION OF THE SKULL WITH ESPECIAL REFERENCE TO THE OCCURRENCE OF MACEWEN'S SIGN IN MENINGITIS AND CEREBRAL DISEASE OF CHILDREN.

BY HENRY KOPLIK, M.D.,
New York.

During the past two years I have been particularly interested in percussion of the skull in children suffering from various forms of meningitis. I was led to this train of thought especially by the writings of MacEwen. In his treatise upon the pyogenic infective diseases of the brain and spinal cord, MacEwen discusses the percussion note obtained over a distended lateral ventricle of the brain, speaking especially of the base of the brain and those diseases, such as tumors, in which an increase of fluid in the ventricle caused by an occlusion of the veins of Galen, or otherwise, produces an altered percussion note over the ventricle.

He says:—"When the lateral ventricles are distended with serous fluid, as would be occasioned by cerebral tumors pressing on the fourth ventricle, or by occlusion of the veins of Galen or otherwise, the percussion note is markedly altered, the resonance being greatly increased Beside the increased resonance, there is an important feature which may be demonstrated:—The percussion elicited at a given spot on the cranium, such as the pterion, varies according to the position of the head. While the person sits with the head upright, the most resonant note is brought out by percussion toward the basal level of the frontal bones and the squamous portion of the parietal. If the patient hangs his head to one side, so that one parietal is placed fairly below the other, the greater resonance is found on percussion of the lower parietal. Reverse the position and the same note is elicited on the opposite side of the head, which is now the lower, the greater resonance being found at that part of the skull nearest the lateral ventricles, and which for the time is at the lowest level.

"These observations tend to indicate that the quality of this note is not dependent on the mere density of the diameter of the cranium, but to a large extent upon the consistence or arrangement of the intracranial contents relatively to the osseous walls. . . . The exact mechanical quality of the note is difficult to describe, but when heard, it conveys the idea of hollowness. One such case, in which the above phenomena were clearly marked was observed to a conclusion. The percussion note was not so clear at first as it

ultimately became, the resonance increasing as the disease advanced."

Taking these remarks as a keynote, I have made observations on every case of meningitis coming under my notice. I have studied, also, the percussion of the cranium in normal infants. MacEwen has studied this note, he says, in over forty children and young adolescents who have had distended ventricles arising from any cause. He says:—"In tumors of the cerebellum it is an aid to diagnosis, and when present with abscess it points to an involvement of the cerebral fossa."

In looking through the literature for notices upon percussion of the head in children, I have found that Betz has studied this phenomenon in normal children. He says that over the cranium, when the bones are thick and formed, there is an empty tympanic note. The percussion is dull over the fontanels where the bones are not formed. There is a sonorous, hollow percussion note where the bones are thin but formed, and a tympanitic note at the back of the skull. He finds a "cracked pot" sound in the normal head, and a parchment-like sound over the large fontanels when the sutures are closed.

These notices of Betz are rather confusing, inasmuch as it is not stated whether the children were recumbent or sitting up when examined. I have tried to simplify a very useful sign of MacEwen as applied to infants and children. MacEwen's sign is the finding by percussion of a tympanitic note over the pterion, the base of the frontal bone and the squamous portion of the parietal bone, that is, over the situation of the horn of the ventricle; but this is not so useful below the age of two years as after this period, when the bones are fully formed and the sutures closed. From the earliest infancy a mild tympanitic note may be obtained either in the recumbent posture or in the position of the head described by MacEwen over the situation indicated, in infants, especially in those who are slightly or markedly rachitic. In some infants in whom the signs of rachitis are very evident, the parietal boss is present to a marked degree and when the fontanels are large this note may be obtained without the least difficulty. I think its presence is due to the cause laid down by MacEwen, an excess of fluid in the ventricles. We know that there is present in many of these cases a very mild degree of serous accumulation in the brain cavities, or hydrocephalus if we might so call it.

In passing from the study of infants to children above the age of two years, the MacEwen sign is especially useful in those forms of cerebral disease, such as tumor of the brain and pachymeningitis, in which there is an accumulation of fluid in the ventricles. In tubercular meningitis I have found the note increased in resonance from the early stages of the disease to that period in which we know that a large accumulation takes place in the ventricle, and upon lumbar puncture I have confirmed to a greater or less extent that there has been not only in the ventricles, but in the subarachnoid space, an accumulation of fluid greater than the normal and present under a considerable degree of pressure.

In cerebral meningitis, and those forms of suppuration in which there is an accumulation of fluid not only in the subarachnoid space, but over the external portion of the brain and cerebellum, the note elicited by percussion is dull; the tympany fails to appear. The failure in these cases to obtain the tympanitic note of MacEwen is due, in my opinion, to the fact that in the suppurative forms of meningitis there is a thin layer of exudate between the dura and the pia mater, thus making the brain substance and the skull cap a continuous medium, as in pleurisy. In fact, one of the most useful differential signs between the tuberculous form of meningitis and the cerebrospinal form (meningococcus) is that, in the tuberculous form of meningitis, this acute accumulation of serous fluid in the ventricles gives us the MacEwen sign, as compared to the rather dull note obtained in other forms of meningitis. I have now under observation an obscure case, in which the MacEwen sign is obtained in all its clearness. The diagnosis of this case at one time was that of cerebral tumor, and this diagnosis was made by a distinguished neurologist.

In conclusion I may say that the MacEwen sign must be accepted with great caution in cases of infants below two years of age. It is one of the most useful signs of fluid in the lateral ventricles of the brain in various forms of meningitis, especially of the tuberculous type, and in tumors at the base of the brain. I have had occasion in 1 case of cerebral abscess, due to otitis and mastoid disease, occurring in a boy six years of age, to find this sign quite marked, so that I can agree with MacEwen as to its value in children as well as adults. It is to be emphasized that in seeking this sign the patient must assume the sitting posture as MacEwen directs; otherwise error is inevitable.

629 Madison Avenue, New York.

I. PARALYSIS OF DIAPHRAGM AND SOFT PALATE (DIPHTHERITIC?): RECOVERY: SEVERE DYSPNEA; RESPIRATION ENTIRELY COSTAL.

II. PARALYSIS OF COSTAL RESPIRATORY MUSCLES: ACUTE HEMORRHAGIC MYELITIS; DEATH IN THREE DAYS: DYSPNEA; RESPIRATION WHOLLY DIAPHRAGMATIC.

III. DYSPNEA: OBSTRUCTIVE AND NOT NASOPHARYNGEAL NOR LARYNGEAL, BUT DUE TO ACCIDENTAL PRESENCE OF AIR IN THE PLEURA, PROBABLY DUE TO THE FAULTY METHODS OF NEEDLE EXPLORATION IN A TUBERCULOUS LUNG.

BY W. P. NORTHRUP, M.D.,
New York.

Intubationists are especially interested in the different varieties of dyspnea. I shall call attention to some unusual types, for I am forced to believe that, even yet, general practitioners will call for intubation in pneumonia without laryngeal obstruction, in retropharyngeal abscess, and I have just learned how urgent diphtheritic croup may be mistaken for severe pneumonia.

Dyspnea of peripheral nerve origin, neuritis presumably diphtheritic, paralysis of diaphragm, is the first type.

Dyspnea of central origin, infectious transverse myelitis and paralysis of the thoracic muscles of respiration, is the second type.

Dyspnea, obstructive and not from stenosis of the nasopharynx, nor yet of the larynx, but due to accidental presence of air in the pleura, probably due to faulty exploration by needle, is the third.

I. PARALYSIS OF DIAPHRAGM AND SOFT PALATE (DIPHTHERITIC?): RECOVERY: SEVERE DYSPNEA; RESPIRATION ENTIRELY COSTAL.

A girl, ten years old, was brought to the Presbyterian Hospital in great prostration and severe dyspnea. There was no history nor evidence of diphtheria, no bacilli grew from nose or mouth culture, no albuminuria. She had previously been in good health, not subject to sore throat. All history was unsatisfactory, and, with reference to diphtheria, negative.

Four days before entrance she complained of pains in the legs. On account of these pains she remained home from school, and her mother relieved them somewhat by rubbing with alcohol. She was not in bed, simply remained at home. There was no history of exposure.

On the day before entrance she complained of pain in the left side, and cough. It was not known that she had a chill. She was put to bed, the breathing became hurried and difficult, presumably on account of the pain in chest, no signs of consolidation.

On entrance these were the notes of her condition:

Pain in one side, dyspnea and cough; much prostration; extremities cold; pulse almost imperceptible; no cyanosis.

The child was poorly nourished, anemic, breathing quietly when undisturbed, and pretty comfortable. She was very dirty and neglected, and the points of her history were in harmony with the general slovenliness of her home surroundings. She could easily have had a mild diphtheria and not been observed.

I was asked to see this case in the Accident Ward. There was something unusual about the case and the house staff wished me to assume the responsibility of receiving her into the wards.

Here is a brief record, after looking backward over the case, and omitting all history irrelevant to the diagnosis: General appearance: She lay on her back, prostrated, quiet and comfortable when undisturbed, very serious looking, apparently just about able to keep up with the needs for breathing when everything was going favorably. At my approach she scowled, turned her head away, shut her eyes and showed signs of dyspnea. She was frothing at the nostrils and mouth corners, there was a moist rattling in the pharynx and trachea, a "death rattle" on respiration, and occasionally a sort of effort at clearing her throat. This we learned to interpret as a futile attempt at cough. It was much like a smoker's clearing of the throat as mucus trails down to his larynx.

Her face was dirty, pale, the malar region flushed. Tongue was moist, the tonsils without exudate, muco-pus in pharynx, no discharge from the ears. There was little or no fever and only a few moist râles in the chest.

The nostrils flared, there was dyspnea, exaggerated, violent costal respiration. The abdomen was empty, the anterior wall lying on the posterior wall. The scaphoid abdomen would have held a little water, and, furthermore, the water would not have

been spilled in respiration. There was no descent of the diaphragm and no heaping up of the abdominal contents. On the other hand, on inspiration, ***the liver and epigastric contents slid up under the ribs,*** were aspirated upwards, carrying the diaphragm with them.

During the first four days and nights in the hospital, the prognosis was grave. The patient could with difficulty be fed. At night, when asleep, the breathing frequently became halting and ceased. A nurse was obliged to be at her bedside all the time. Several times each night the nurse compressed the chest and performed artificial respiration. The child begged to be left undisturbed, "If you'll only let me alone," she said.

The evidences of palatal paralysis were: Difficulty in swallowing, inability to lift the palate (velum) when touched with a probe, inability to pronounce such words as "Rosie," her name. During convalescence she had difficulty, more than all, in saying, "tooth." The latter was such a dismal failure and evoked so much amusement, that the child cried and would not try before company. The whole ward was entertained.

The evidences of paralysis of the diaphragm were: Exaggerated costal movement, moving upward, during inspiration of the liver and epigastric contents, inability to produce explosive cough.

The reader may simulate the sound of her peculiar cough by suddenly contracting the abdominal walls leaving the diaphragm, palate and larynx lax.

There was slight internal strabismus.

Urine was normal; no albumin.

There were never any evidences of diphtheria, but it is supposed she had an undiscovered mild diphtheria, which untreated, ran its full course and resulted in toxic neuritis, with consequent paralysis, ocular, palatal and diaphragmatic.

As to the subsequent course, there never was any fever, or albuminuria, the diaphragm began to show a little action on the fourth day, and in ten to fourteen days was in full function.

The soft palate gradually improved, but was not in full function for a month.

Finally the girl left the hospital quite well. "Rosie" and "tooth" remained her test words to the last, and only at the end of a month was she able to acquit herself favorably on these.

II. PARALYSIS OF COSTAL RESPIRATORY MUSCLES: ACUTE HEMORRHAGIC MYELITIS; DEATH IN THREE DAYS. DYSPNEA—RESPIRATION WHOLLY DIAPHRAGMATIC.

The patient was a twin girl, three and one-half years old, living in Stamford, Conn., tended by Drs. Rogers and Samuel Pierson.

For one week the girl had an ordinary cold, with some bronchitis, and was in bed the early part of that time.

Dr. Rogers considered her, after four days, nearly well, and discontinued his visits. While quietly playing at her father's knee the child suddenly fell to the floor, and was unable to rise. No pain.

On being lifted up, her legs were powerless, "hung down," as the father observed, "like two empty stockings." She was put into bed, vomited and continued to do so many times. An hour after attack pulse was 120°, respiration 30°, temperature 104°.

About twelve hours after first attack, Dr. Pierson saw her in consultation. Temperature then was 105° F., pulse 140°, regular and full, respiration 40°. Sensation absolutely abolished below the waist. Vomiting severe, prolonged and frequent. Lungs negative, neck somewhat resistant.

Twelve hours later, or twenty-four hours from the attack, I saw the case, with the two gentlemen mentioned. At this time the temperature had fallen to 100° plus. Vomiting was less, but her other symptoms were more marked. Respiration was labored and abdominal. Her appearance suggested shock. She was very pale, relaxed, her face mask-like, eyes half closed. Dyspnea marked. Her abdomen was distended and heaving violently; her thorax did not expand at all. Nostrils flared.

There was slight stiffness of neck, but no other evidences of meningitis. No paralysis of face or tongue. Spleen not feelable. There was paralysis of the muscles of the thorax and paraplegia—*i. e.,* absolute loss of motion and sensation in lower limbs, loss of patellar reflexes, retention of urine, incontinence of feces.

The ability to phonate was present till death. Pulse continued regular, respirations were always regular, becoming more and more shallow. Patient became very restless, tossing head from side to side, and died suddenly, forty-five and one-half hours after the first attack.

DYSPNEA: The nostrils flared, the thorax was still, the abdomen heaved. The respirations were labored, wholly abdominal,

regular. The abdomen being distended, made its heaving more marked and a most striking phenomenon to witness.

Another observation, and never explained, was this: no air entered the left lung anteriorly. There was no dullness—simply no sound of air entering. On the other hand, air entered the other lung violently, making an exaggerated sound. There were a few râles posteriorly, and a declining temperature, no evidence of consolidation or fluid. Heart in normal position. Digestion good.

Our diagnosis has been submitted to expert neurologists and stands corroborated as follows:

Acute infectious transverse myelitis, the lesion affecting the dorsal and lumbar portions. The infection possibly influenza. The month was March and influenza was said to prevail in the vicinity.

The whole course of the disease was so rapid that no blood studies could be carried out, and there was no autopsy.

III. DYSPNEA: OBSTRUCTIVE AND NOT NASOPHARYNGEAL, NOR LARYNGEAL, BUT DUE TO ACCIDENTAL PRESENCE OF AIR IN THE PLEURA, PROBABLY DUE TO FAULTY METHODS OF NEEDLE EXPLORATION IN A TUBERCULOUS LUNG.

While witnessing an operation in the amphitheatre of the Presbyterian Hospital, I was asked to examine a young child just brought in for empyema, and if I considered it an operative case, to send it to the table at once.

I found a child of two years sitting upon the bed, laboring for breath. It was pale, anxious, wholly absorbed in the business in hand, namely, getting sufficient air. Its mouth was dry and it was trying to moisten its lips with a dry tongue. It was panting, grunting, preoccupied. It seemed rather an imposition to examine it and worry it.

The heart was much displaced to the left and downwards. Its right chest was dull, front and back, near the median line up and down. There was over this area diminished breathing, moderate dullness, bronchial voice and breathing indistinct and distinct. However, in the lateral half of the chest, a large lenticular displacement by air was observable. This centred in the axillary line and about a needle prick seen in the skin.

From this needle site the father reported that the doctor had aspirated pus and had consequently sent the case to the hospital for operation. It was obvious that the child would not live long unless

relieved. Air was surely present and crowding the heart over and down. Pus was probably present. The child was anesthetized lightly, the chest opened and air escaped freely, but no pus. Everywhere were adhesions and consolidated lung, but no pus.

The operation was short, the heart returned partly to place, and the case was returned to the ward to linger, as it was believed, a little while and probably die. We felt that there must be an abscess either mediastinal, between the lobes or near the root, or that there must be mediastinal tuberculous masses. Dr. McCosh, the operator, could find neither abscess nor masses. The condition of the child warranted but the briefest operation.

At present writing the case is still in the hospital, running a low temperature and comfortable. The diagnosis now is tuberculous consolidation, with probably cheesy masses about the roots and within the anterior mediastium.

It is unusual, in my experience, to have any kind of a mishap from exploration by needle. In the present case we have no knowledge of the manner of using the needle and what led to the accumulation of air. From my experience and observation I should say, no accident can happen, barring adding another infection. Moderate cleanliness will avert that.

Finally, the dyspnea which constituted the most prominent and the most urgent symptoms was due to the air in the pleura, crowding and embarrassing lungs and heart. Releasing this air caused instant relief and lasting freedom from dyspnea.

This I have called an obstructive dyspnea.

P. S.—Three and one-half weeks later the child died, and partial autopsy throws this light upon the case. At the time of operation there was a deep tuberculous softening, perhaps abscess, at the root of the lung. The needle possibly reached this and allowed air to escape into pleura. At the time of autopsy the whole central portion of the lung was excavated and drained through operation wound and bronchi. The lung resembled the cavity of a cocoanut, with thin wall all about originally adherent, everywhere.

DISCUSSION.

DR. HOLT.—All of Dr. Northrup's cases are very interesting. His first case I have seen paralleled where the symptoms came on late in diphtheria and where no treatment, or late antitoxin, had been given. I have never seen anything exactly like the third case.

DR. CRANDALL.—Dr. Northrup's third case is interesting and I have seen a case that perhaps explains it. I was called to aid a physician who by mistake had reversed his aspiration pump and injected air into the pleural cavity. The patient was in an extreme condition of dyspnea and cyanosis, but was relieved by aspiration.

DR. ABT.—Greater care should be exercised in making aspirations. I believe that in unskilled hands harm can be done. The needle used is often unnecessarily long and is introduced at too great depth. I have frequently watched hospital internes aspirate the chest, who used a needle 3½ or 4 inches long, and plunged it deeply into the thorax. If a long needle is used, it should be guarded by winding sterile cotton about it so that not more than 1 or 1½ inches of the needle penetrates the thorax. It is preferable to use a short needle, which should be of somewhat wider calibre than those ordinarily used. If all the cases of accidents from hemorrhage, infections, broken needles, etc., were at hand, a large list would be furnished. I recall seeing 1 case in which peritonitis developed, the infection being carried from the pleural cavity into the peritoneum along the track of the needle.

DR. CAILLÉ.—I have seen pneumothorax from puncture on two occasions and it was an unfortunate accident. I reported to the Society a series of such cases years ago. It happens when the point of the needle enters the lung and too many lateral excursions are made with the needle.

DR. NORTHRUP.—I believe I have seen about everything that can happen from the use of the needle, but I have never seen any serious harm done.

BRONCHIAL ASTHMA IN INFANTS AND CHILDREN, WITH AN ANALYSIS OF FORTY-THREE CASES.

BY LINNÆUS E. LA FÉTRA, M.D.,

Instructor in Pediatrics, College of Physicians and Surgeons (Columbia University); Chief of Department of Pediatrics, Vanderbilt Clinic; Assistant Attending Physician to the Babies' Hospital, New York.

Spasmodic or bronchial asthma is not a rarity, even in infants, and is fairly frequent in children, the number of cases met with increasing with the years of the patients. During the past two years there have come under my notice 43 cases of asthma in infants and in children under twelve years of age; 38 of these cases were under my care at the Vanderbilt Clinic, and the other 5 occurred in my own practice. As to the relative frequency of cases of asthma, I find that they make up slightly more than 1 per cent. of the whole number of patients. During the past twenty months there were 3,460 new patients under twelve years treated in the Children's Department of the Vanderbilt Clinic; of these thirty-eight had attacks of spasmodic dyspnea along with physical signs sufficiently characteristic to be classed as asthma. During the same period and among the same patients there were 851 cases of bronchitis, almost 25 per cent. Thus there would be, on the average, 25 cases of bronchitis—not including any form of pneumonia—to 1 case of asthma. In addition, it is of interest to note that there were during the same period 2 cases of emphysema and 13 cases of emphysema with bronchitis, which were entirely free from asthmatic attacks.

Age of Incidence.—Characteristic symptoms appeared at birth in 3 cases; from birth to three months in 2 cases; from three to six months in 3 cases; from six to twelve months in 3 cases. This makes a total of 11 cases occurring during the first year of life; of these 11, 8 were females and 3 males. During the second year of life there occurred 8 cases, 6 males and 2 females; from two to five years, there were 9 cases, 8 males and 1 female; from five to twelve years, 15 cases, 10 males and 5 females. No patients over twelve years of age were treated. It will be noticed that

only during the first year was there a preponderance of females over males; after that the males exceed the females, and in the period from five to twelve years the ordinary ratio of adult asthma is attained, there being a marked preponderance of the males. In the whole number of 43 cases, there were 27 males and 16 females. During the first year of life it may be that the smaller size and weaker resistance of the females has something to do with their greater susceptibility, either to asthma itself, or to the bronchitis which so often precedes the asthmatic attacks.

Hyde Salter,* in his classical monograph, among 225 patients of all ages, gives 11 cases as having begun during the first year of life, his youngest patient having been fourteen days old at the time of the first asthmatic seizure. From one to ten years Salter records 60 cases, and from ten to twenty, 30 cases. Goodhart,† among 123 cases of all ages records 50 cases under ten years old, 31 males and 19 females; his youngest patient was three and one-half years old.

Length of time the asthma has persisted.—At the time the patients came under treatment the disease had existed less than three months in 9 cases; from three to six months in 5 cases; from six to twelve months in 4 cases; from one to three years in 9 cases; from three to six years in 5 cases; over six years in 3 cases. The most persistent case was one that had existed for nine and one-half years in a boy eleven years old. The most distressing one occurred in a colored girl of thirteen years, who had been unable to sleep lying on her back for seven years. If the ages of the patients at the time they came under treatment are combined, the sum is 282 years; if the periods of symptoms are combined, the sum is eighty-seven years; thus, the average duration of asthma in these 43 patients was almost one-third of their lives.

Race.—The nationality of the parents or the birthplace of the patient was given as the United States in 16 cases; German, 8; Swedish, 2; Dutch, 1; Irish, 6; United States, colored, 2; Hebrew, 9 (of these 3 were Germans, 1 an Austrian and 5 were Russians). There was not a single Italian and only two negroes; this was remarkable, since one would have expected rachitis to be a very important predisposing factor, as it is in cases of spasmodic croup and bronchitis.

Family History.—The family history was asthmatic or rheu-

* The Pathology and Treatment of Asthma.

† Allbutt's System.

matic or tuberculous or neurotic in many cases, but the answers were not sufficiently reliable to be suitable for statistics. Authorities are agreed, however, that the tendency to asthma is transmitted in many instances by asthmatic ancestors—in 16 per cent. of Berkart's cases.

Abnormalities of development were present in several cases, especially where the symptoms began at birth or very early in the first year. There was pulmonary atelectasis in 2 cases, an enlarged thymus gland in 2 cases, pigeon breast in 1 case; two of the patients were prematurely born, and one was of the mongolian type of idiocy.

The relation to adenoids will be spoken of later.

Previous Personal History.—Twenty patients had previously suffered from bronchitis, several of these from a bronchitis that was a complication or sequel of measles or whooping-cough. There had been bronchopneumonia in 7 cases, one of these part of a grip infection; so that altogether 27 out of 43 patients had had some respiratory inflammation preceding the attacks of asthma, Rickets were present, but nearly always in mild form, in 8 cases. Only one of the patients had suffered from chorea, and none from migraine or epilepsy.

Cause of the Attacks.—Immediately preceding the attacks, or coincident with them, and therefore assigned by the parents as a causative factor, there was bronchitis in 14 cases; bronchopneumonia in 1 case; pneumonia in 2 cases; gastric indigestion, a drive against a cold wind, a fall down stairs and an attack of malaria, each in 1 case. Thus, in more than half the cases no cause was suggested. Each of the above circumstances has been known to cause attacks. In India, according to Goodhart, malaria is one of the usual causes.

Periodicity and Severity of the Attacks.—The attacks were said to occur daily, or with no stated intermission, in 8 cases; in the others the periods varied from two weeks to "every winter" or "every summer." The attacks were worse by day in only 2 cases; all the rest were worst at night; or, in the case of three infants, whenever they were lying on the back; one of these patients had a large thymus which might explain the access of the paroxysm when supine. Three cases were distinctly worse in hot weather; the others were generally worse in winter.

Complications.—Emphysema alone was associated with asthma in the cases of 6 boys and 4 girls; 5 of these patients had the

square or barrel chest, their ages being two years, five years, five years, nine years and twelve years, respectively. The youngest patient of this group, a baby boy of two years, had had attacks noted as constant for three months following a bronchitis; along with the emphysema there was a tendency to urticaria.

Emphysema with bronchitis was present in 2 girls and 3 boys. Only one of these patients had a barrel chest; he was a little boy three years old who had had asthmatic attacks for over a year following bronchitis; large adenoids were also present. The shape of his chest and its movements were typically emphysematous.

Abnormalities of the nasopharynx were associated as follows: Congenitally high-arched palate (without adenoids) in 1 case; deviated septum in 2 cases; adenoids or adenoids and large tonsils in 20 cases. There was a lingual tonsil in 1 case. It will be seen that 47 per cent., or nearly half the cases, suffered from adenoid growths. The presence of adenoids in so large a proportion of asthma cases is very significant, for probably not more than 30 per cent. of New York dispensary children have adenoids sufficient to cause symptoms.

Symptomatology.—During the attacks, whether separated by long periods, or occurring every few hours, there is the same wheezing, whistling breathing, and dypsnea, all the accessory muscles of respiration being called into requisition. The patients are usually relieved by sitting up or by having the shoulders raised and the elbows braced so as to give better support to the muscles passing from the head, arms and shoulders to the chest. There may be cyanosis. In infants and young children there is, at each inspiration, sinking in of the suprasternal and supraclavicular fossæ and of the rib insertions of the diaphragm below; while in older children or in protracted cases the whole chest moves upward with even less expansion. Toward the end of a paroxysm the chest may become temporarily barrel-shaped; but after the attack has passed the emphysema disappears and the chest resumes its normal form. Repeated or prolonged attacks produce a permanent deformity, as has been noted in the above analysis. The respiratory rate in infants may be anywhere from 44 to 80 per minute, and with the alæ nasi dilating beginning bronchopneumonia is suggested.

Palpation reveals bronchial fremitus in most cases.

Percussion early in the paroxysm elicits a note that is some-

what dull; toward the end of the attack the note may be hyperresonant.

Auscultation in the uncomplicated cases reveals dry squeaking or whistling, along with some musical or sonorous râles, both during inspiration and expiration; the inspiratory sounds are the louder, while the expiratory sounds last longer. These sibilant and sonorous râles may be simultaneously heard all over the chest or at different parts in succession, coming and going during the attack. Often the pandemonium of the chest can be heard at a distance of 20 or 30 feet, even in an infant. Notwithstanding all this adventitious noise the breath sounds themselves are actually feeble, very little air entering the chest. Toward the end of an attack, and in cases complicated by bronchitis, there are subcrepitant and mucous râles.

Cough, if present, is short and dry, except toward the end of an attack, when it is looser. In older children there is some expectoration. I have never examined the sputa for the characteristic crystals and spirals.

Spontaneously, or after the administration of some sedative, the respiration becomes less and less noisy, and less rapid. The sibilant râles are replaced by larger sonorous and mucous râles, and soon only a few large moist râles and sonorous breathing remain to signalize the storm that has past. The hyperresonant note disappears and the chest resumes its usual shape and normal respiratory rhythm. The temperature in uncomplicated cases is rarely above normal, except in infants, where it may rise to 100°F. and 101°F.

Eosinophilia.—The leukocytes are usually, but not always, increased, as in bronchitis, but a differential count of the white cells shows what does not occur in bronchitis, a constant and usually marked increase in the number of poly-eosinophiles. The cases examined for me at the Vanderbilt Clinic by Dr. Ira Wile, the clinical pathologist, showed an eosinophilia from 6 to 18 per cent. Cabot reports in adults a mean eosinophilia of 7 per cent. in a range from 0 to 53.6 per cent. This eosinophilia is greatest for any given patient at the height of the attack; it disappears in the intervals, but in the subacute cases a low grade eosinophilia persists. Thus the differential count of the leukocytes is of diagnostic and prognostic value, so far as the attack is concerned.

Diagnosis.—Asthma in infants may simulate bronchopneumonia or a fine bronchitis in their early stages when there is little

or no secretion, rapid labored breathing and feeble respiratory murmur, but the low temperature, and the dry whistling râles that flit from one part of the chest to another will suggest the real diagnosis. If such symptoms follow or accompany gastric indigestion, urticaria or attacks of sneezing, asthma is all the more probable. The existence of eosinophilia, together with the speedy recovery from the attack, and later recurrences will confirm the diagnosis.

If one keeps in mind Holt's classification of the symptoms into four groups, the cases are less apt to be mistaken. In my series of cases, however, there was not one of clear-cut hay asthma, though three of the patients always began by sneezing.

Pathology.—The patient does not die of asthma itself; therefore its pathology is not known. Exception must be made in the cases of very young infants, in whom the disease may prove fatal, though uncomplicated.

Pathogenesis.—All clinicians are agreed that the peculiar type of breathing is induced by a narrowing of the lumen of the bronchial tubes. How this stenosis is brought about has given rise to warm discussion. At present only two theories seem worthy of consideration, since the cases can all be explained on one or the other hypothesis.

I. The first and generally accepted theory is that asthma is due to *spasmodic contraction of the smooth muscle fibres of the smaller and medium sized bronchial tubes;* this contraction may affect successive parts at different times, or all of the bronchial tubes at the same time, the tendency being for relaxation in one part while another part is in contraction. For a long time there was no experimental support of Hyde Salter's masterly championship of this theory; but in 1891 Lazarus* showed that electrical stimulation of the nasal mucous membrane will produce a distinct increase in intrabronchial pressure by reflex contraction of the bronchioles through the efferent fibres of the vagi.

Within the last two years Brodie and Dixon,† by a very interesting series of experiments, demonstrated the truth of this theory. They recorded the volume of air entering and leaving the lung at each respiratory act by using an oncometer upon an

* *Deutsche med. Woch.*, 1891, Vol. xvii.. p. 852.

† Brodie and Dixon. Transactions Pathological Society, London, Vol. liv., 1903, p. 17.

isolated lobe of dog's lung. Aeration was kept up artificially and then electricity and various drugs were used to affect the calibre of the bronchi. Stimulation of the vagus caused marked diminution of the volume of air entering and leaving the lung, owing to contraction of the bronchial muscles. They found, too, that the vagus contains also broncho-dilator fibres. They failed to produce any reflex constriction by excitation of a cutaneous nerve, or by excitation of the stomach and intestines. The most important and powerful constrictor reflex was obtained by stimulation of the nasal mucous membrane, particularly the upper and posterior part of the nasal septum. Of drugs, muscarin, pilocarpin, physostigmin, digitalin and carbon dioxid gas produce marked bronchial constriction. Solutions of Charcot-Leyden crystals intravenously injected proved quite harmless. Atropin, hyoscin and hyoscyamin paralyze the vagus endings and cause broncho-dilatation if constriction be previously present. Lobelia causes marked, and morphia only slight, dilatation.

Another argument they advance for bronchial spasm as the cause of the stenosis is that the sudden onset and rapid disappearance of the attack are exactly similar to the common course of prolonged contraction of involuntary muscle. Moreover, a dose of muscarin can induce a typical attack of asthma in an animal; further, such an attack can be cut short almost instantaneously by injection of atropin—an impossibility if the stenosis were due to turgescence of the mucous membrane or to an exudation. Again, the relief patients experience from administration of atropin, lobelia and morphin, which are broncho-dilators, points to same conclusion.

As to mode of production of the bronchial constriction in asthma, they believe it to be due to irritation of the nucleus of the tenth pair of cranial nerves in the medulla by reflex stimulation—as from the stomach after an indigestible meal or from the nasal mucous membrane after irritation by pollen, inflammation, etc. Sneezing cases, they argue, are the best ones for cure by operations on the nasal mucous membrane.

Brodie and Dixon conclude that the reflex contraction of the bronchi is protective, a mode of defence to the organism, to prevent the entrance of irritating substances, whether particulate or molecular, into the respiratory tract. In asthmatics there is an exaggeration of this normal protective reflex.

Undoubtedly nearly all the cases in infants and a majority of

the adult cases of asthma are explained by the above theory of contraction of the bronchial muscles.

II. The second theory is that the narrowing of the bronchial lumen is due to *vasomotor tumefaction, turgescence or paresis*—a sort of angioneurotic edema or urticaria of the bronchial mucous membrane. So far as I know this theory is supported not by experimental evidence, but by analogy and by the clinical results of certain forms of treatment. Comparison is made with the turgid state of the nasal mucous membrane brought about by a draught, by irritating vapors, by powdered ipecac, or by pollen in a hay fever subject, and the assumption is that the same sort of vasomotor swelling takes place in the bronchial mucous membrane. The association of asthma with urticaria and the analogies of the two conditions have been emphasized by the late Dr. F. A. Packard,* who reported 5 cases where asthma was replaced by urticaria. Several cases are reported by Crouslé,† in one of which the asthma that followed eating mussels was relieved at once when urticaria appeared on the skin; in another case, a causeless coryza was apparently connected with an outbreak of hives, and the urticaria was substituted by an attack of asthma.

In two of my series of cases the asthma and the urticaria seemed to replace each other. In another patient attacks of eczema seemed to replace the asthma, and *vice versa.*

Again, the results of the injection in asthmatics of the vasoconstrictor principle of the suprarenal gland, as reported by Bullowa and Kaplan, would indicate that in some cases, at least, vasomotor paresis or turgescence is a cause of the narrowed bronchial lumen.

Moreover, in four of my series the use of adrenalin or suprarenal extract, either internally or in the nose, was followed so quickly by marked relief of the symptoms that it would seem fair to assume vasomotor paresis in those cases. The wide use of adrenal sprays in hay asthma is also in favor of this theory.

Against this interpretation of the local use of a vaso constrictor it may well be urged that by closing up the vessels of the nasal mucosa the particulate exciting cause is prevented from acting on the nasal branches of the trifacial nerve, and so the reflex spasm

* Urticaria of the mucous membranes, especially in connection with asthma. Transactions American Pediatric Society, Vol. xi., p. 92.

† Thèse de Paris, 1889.

is not produced. No such objection, however, can be made in case of the hypodermatic use of adrenalin.

So far as this second theory is concerned, I think we can safely say that some of the cases of bronchial asthma are produced by vasomotor turgescence.

Etiology.—Patients that suffer from asthma have a peculiar susceptibility to irritations of the upper respiratory tract and to all reflexes physiologically connected with the pneumogastric nerve, and probably the thoracic sympathetic nerves. This susceptibility is analogous to the idiosyncrasy possessed by some people to strawberries, shell-fish and woolen clothing—that is to say, it is an abnormal response to a normal stimulus; it is an exaggeration of the activity of a normal protective reflex. We see it acting normally where there is contraction of the bronchi to prevent the entrance of irritating vapors, and it is present, also, in certain stages of bronchitis.

The constitution may be asthmatic by reason of asthmatic, gouty or neurotic inheritance, or because of frequent attacks of bronchial and pulmonary inflammations, especially those complicating measles and whooping-cough. Eighty per cent. of Salter's cases followed pertussis. These inflammations lower the tone of the general health and heighten the irritability of the nerves involved. Or, the susceptibility may be strictly local and due to adenoids, polypi, hypertrophied turbinated bones, elongated uvula, lingual tonsil, enlarged bronchial lymph nodes or a persistent thymus gland.

Exciting causes of the attack, aside from bronchitis or a "cold," are difficult to discover in infants and children. In contrast with adults, only one or two seemed to be affected by dust; 2 cases were worse in summer and may have been due to vegetation; in 3 cases the attacks were preceded by sneezing, so that these may have been also hay-fever cases. Sudden change of the weather from hot to cold, or *vice versa,* seemed to have more effect than damp weather, unless there was bronchitis. Warm damp weather seemed favorable in one instance.

In view of Prof. Dunbar's experiments with the toxins and antitoxins of hay fever, and in view, also, of the eosinophilia, which occurs likewise in cases of trichinæ, tape worm and echinococcus infection, it is rational to believe that in all cases some toxin has been manufactured in the body or absorbed from the respiratory tract.

Treatment.—With full knowledge of the predisposing and usual exciting causes, and of the two methods by which stenosis of the bronchi is produced, a careful study of the patient's history and physical signs will suggest in nearly every case the rational and successful line of treatment. Predisposing and exciting causes should be removed wherever that is possible; if they cannot be removed they can frequently be modified; in the case of unfavorable climatic, aerial or locality conditions, the patient should be removed from the cause. The attacks themselves are to be treated on the basis of their pathogenesis; subsequently, the patient's general condition is to be toned up by hygienic and dietetic measures, and the respiratory neuromuscular mechanism rendered less sensitive by sedatives at first, but later by alteratives and nerve tonics.

Prophylaxis.—In the case of infants and children who have never had an attack, but whose ancestry is asthmatic, as well as in the case of asthma subjects, prophylaxis may do much to diminish the probability of subsequent attacks. The chief predisposing causes are bronchitis and pharyngeal adenoids. Attacks of nasopharyngitis and bronchitis are never to be lightly regarded in infants, and active treatment should be carried on in each case to shorten the disease and so prevent the respiratory mucous membrane from becoming over-sensitive; it is particularly important not to let the colds "hang on" in those who have an asthmatic, gouty or rheumatic heredity, nor to let whooping-cough and measles be complicated by a prolonged bronchitis. The prevention of taking cold involves the whole hygiene of the child; particularly seeing that the clothing is not so heavy as to keep the child in a constant bath of perspiration, and attending to the matters of daily cold sponging or bathing, daily exercise out of doors, and proper airing of the nursery by day and by night. Parents are apt to make hot-house plants of such children—and all because the physician has insisted, rightly enough, that the child should not be allowed to catch cold.

Removal of adenoids and enlarged tonsils greatly diminishes the liability to asthmatic attacks. The operation may be followed in a small percentage of cases immediately, in the majority of cases after a few weeks, by a cessation of the attacks. In my series of cases, 8 gave indisputable evidence of the value of the operation; but in several others, older cases, there was no perceptible improvement.

Hypertrophic rhinitis, polypi, lingual tonsil, or deviated septum should receive the proper treatment, severer measures being used if cleansing and the application of adrenalin does not produce visible improvement. A regular toilet of the nose, as practised by Jacobi, should be established, an alkaline saline solution being used night and morning. For enlarged bronchial lymph nodes or persistent thymus gland, arsenic and the syrup of iodid of iron should be given.

The removal of the exciting causes, while of the first importance, is often impossible because they are not recognized. Where the attacks accompany gastric indigestion or urticaria, the offending articles of diet can be excluded, or the whole dietetic *régime* modified. Where the attacks are due to vegetation, certain odors, animal emanations or dust, the cause and the patient should, of course, be put apart. Cats and horses have proved exciting causes, and should be eliminated in every case. A high dry climate in winter, or the woods in summer, may be the proper environment for those who have attacks in the city. Usually, however, the city is a good place for hay-fever patients.

The removal of the local cause frequently does not eradicate the habit when this has become established, and then other measures, hygienic and medicinal, are required.

Treatment of the Constitution.—It is of extreme importance not to relax treatment until recurrences of the attacks are prevented, inasmuch as emphysema, chronic bronchitis and permanent changes in the chest occur in protracted cases or in those with frequent attacks. All the measures mentioned under *prophylaxis* should be employed, and certain additional drug treatment is desirable.

To keep the respiratory membranes well toned up during the winter, cod-liver oil, in connection or in alternation with tonic doses of quinin, is of great value. In place of quinin, the combination of guaiacol and quinin, known as guaiaquin, in doses of two grains three times daily, has been employed with advantage. Arsenic also may be used in place of or in alternation with the quinin preparations, both for its effect on the blood and on the nerves of the respiratory mucous membranes, diminishing their excitability by raising their tone.

For cases of long standing nothing takes the place of the iodids. The iodid of sodium is well borne by children when given with liquid peptonoids, trophonin or essence of pepsin. It is

probable that the employment of Dunbar's pollantin, and of other hay fever antitoxins will prove beneficial in those patients who are hay asthma subjects.

Rheumatic children and those subject to eczema or urticaria should have a carefully regulated diet, and their liver should be kept active by the use of phosphate or salicylate of soda, a series of calomel being administered once a month or oftener.

Treatment of the Attack.—The great majority of the attacks are due to spasm of the bronchial muscles. Accordingly, measures are used to relax the spasm. Of first importance are those that can be administered by inhalation. In infants, where the attacks closely resemble capillary bronchitis, the effect of steam impregnated with creosote (20 drops of creosote being poured into a pint of water boiling in a croup kettle) is very satisfactory in relaxing the spasm. In older children, the nitrate papers, with or without stramonium, are efficient. The proprietary asthma cures do very well, consisting of nitre and stramonium leaves for the most part; but in infants the fumes are at times irritating. Where the spasm is intense and the dyspnea dangerous, chloroform may be needed to permit air to enter the lungs. I have had no experience with amyl nitrite and pyridin; they are said by some observers to be good, though pyridin is uncertain in its action.

As soon as the infant can swallow, a dose of tartar emetic, 1-100 grain, with ipecac 1-20, should be given, along with 1-100 or 1-50 grain nitroglycerin; this dose is repeated every half hour for two or three times, or until relief is procured. This plan is particularly good when the case resembles a bronchitis of the finer tubes.

Another method which I have used with great benefit is to give atropin in doses of 1-2000 grains every two hours until the face flushes; the effect of the atropin seems to come in about two or three hours. Infants vary in their susceptibility to the flushing, but usually a baby of two years can take two or three of the above doses before the face gets red. At times I have found the paroxysm pass off and the sibilant râles disappear within an hour after the first dose. After the attack has passed off the atropin is given twice or three times a day for several days.

If the attack is one accompanied by gastric indigestion, the stomach should be emptied at once, preferably by a dose of syrup of ipecac.

For long standing cases and those in older children, the old

emphysema mixture has been used for years with success. The composition is as follows:

℞—Potassii iodidi	grs. xx	
Morphinæ sulphatis	gr. ⅓	
Tinct. belladonnæ		♏xx
Spts. ætheris comp.		f℥iii
Aquæ	qs. ad	f℥ii

It is given in dram doses every three or four hours, and is especially of value when there is complicating emphysema or bronchitis.

On the theory that the attack is due to vasomotor paresis or turgescence, vaso constrictors should be used. I have never given adrenalin chlorid hypodermatically to children; the dose for adults is from 3 to 10 minims of the 1-1000 solution deeply injected. I have used suprarenal extract solution and the adrenalin chlorid solution for instillation into the nose in a few cases with marked benefit. The instillation into the nose may not act by vaso constriction of the bronchial blood vessels; but its effect has been beneficial in several cases.

One patient, a colored girl thirteen years old, had had asthma for seven years; she has constant asthma emphysema with a typical barrel chest, and has been unable to sleep lying down for years, and is shown by Dr. Holt to his classes each year. This spring she was treated by instillation of ten drops of suprarenal solution into the nose every two hours for six doses each day. In dispensary practice the adrenalin chlorid solution is too expensive, so this solution of the powdered extract of the gland can be used instead. The first night, after this treatment was begun she slept all night lying down and the second day afterwards the sibilant râles had almost entirely disappeared; the girl said she never had been so well. It is too early to call the case a cure; but the improvement is remarkable.

Prognosis.—Except in very young infants and in those with enlarged thymus gland the outlook for asthma patients is good in inverse proportion to the time the symptoms have existed and to the frequency of the attacks. In infants the elasticity of the lung and its recuperative power are great; bad nerve habits are more readily overcome. If there is a removable predisposing cause, or if the exciting causes can be removed, there is little doubt of a cure.

In older children, where no cause can be discovered, change of residence to a place where they do not have asthma for a year or two will usually cure them of the tendency.

Altogether the prognosis is favorable if the cases can be put upon proper treatment before emphysema and the barrel chest have developed.

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DISCUSSION.

DR. JENNINGS.—At the meeting of the Society held in Boston, I reported a case of autointoxication in which the chief clinical manifestation was attacks of coma, one in particular being severe and prolonged. The clinical picture has changed during the last year and now, instead of the coma, the patient has attacks of asthma. These attacks of asthma have apparently replaced the other clinical phenomena.

This child came into my office a short time before for an examination of her nasal chambers. To facilitate examination I sprayed the nasal passages with a solution of adrenalin and this set up a severe attack of asthma. During the past two years her attacks have been relieved by heroin in $^1/_{50}$ grain doses administered every half hour until four to six doses have been taken.

DR. HOLT.—These little patients, six or eight years old, seen in private practice, are pitiable cases. It seems to me that for a radical cure of the asthma the one important thing is a change of climate, and this is the only thing that can really be relied upon. I have always felt that any child who continued to have asthma until ten or twelve years old would probably suffer for life; therefore, when I meet with a severe type in younger children I advise that they be taken to a climate where attacks do not occur; although such a place cannot always be found. In many of these patients the general nutrition is at fault and they should be given cod-liver oil, particularly in the winter and spring, and they should be kept in the open air. Most children who have asthma in winter and spring are sufferers from repeated attacks of bronchitis, the beginning often being whooping-cough or measles. I believe that general practitioners and specialists, also, should take these cases of asthma more seriously, and insist upon the parents doing all possible for the small patients, and before the asthma became an established condition.

DR. CRANDALL.—All know how hopeless and how very serious these cases are when once they get a headway. If anything is to be done at all it should be done early, and particularly after

measles. I presume that more of these cases are seen after such epidemics. I recall one family in which the mother was asthmatic and, later, the children showed a tendency to the same trouble. After measles the children were sent south as soon as possible.

DR. NORTHRUP.—I am surprised regarding the frequent occurrence of asthma, and I would protest against people talking about "outgrowing" the asthma.

DR. CAILLÉ.—When I was in Hamburg last year, I was the guest of Dr. Dunbar, who showed me what he could do with the pollen poison and its antitoxin. He was himself a fever patient. He took the pollen poison in solution and instilled it into the eyes of us both. My own eyes were not affected, but Dr. Dunbar's became blood red within fifteen minutes. He then took the antitoxin (pollantin) and placed it in his eye; within half an hour all evidences of vasomotor disturbances had disappeared. It was a striking experience and showed the efficacy of pollantin.

A CASE OF PULMONARY OSTEOARTHROPATHY LIMITED TO THE TERMINAL PHALANGES IN A CHILD; WITH A BRIEF CONSIDERATION OF THE RELATION OF OSTEOARTHROPATHY TO THE CLUBBED FINGERS OF CHRONIC HEART AND LUNG DISEASE.

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In 1890 Marie first described, under the title "De l'Osteoarthropathie Hypertrophiante Pneumique," the skeletal disease characterized by thickening of the long bones in the vicinity of joints, with a peculiar clubbing of the finger ends, similar to, yet not exactly identical with, the ordinary clubbed fingers of chronic heart and lung disease. Because, in the cases described by him, the condition was secondary to some pulmonary affection, he appended the adjective pneumique, or pulmonary, thus adding to the length of a title already sufficiently cumbersome. Independently, and almost simultaneously (1889 and 1891), v. Bamberger reported a series of similar cases, which he thought depended upon hypertrophy and sclerosis of the bones. Since the appearance of these papers, many writers have discussed the affection, and many cases have been reported. The lengthy and somewhat misleading title, in spite of the appearance of several substitutes, has remained; probably because none has as yet been suggested that will adequately describe the multiple clinical features of the affection. Of all the substitutes, the name proposed by Arnold, secondary hyperplastic ostitis, seems the most appropriate, because the bone changes occur in other than pulmonary disease, and the joints usually show no essential change. Osteoarthropathy is always a secondary disease; it has followed cystitis, pyelonephritis, syphilis, dysentery, chronic jaundice and other affections, while there are a few cases on record in which no

primary disease could be discovered (Decloux et Lippmann, Stevens, *et al.*). By far the greater number, however, have occurred in connection with affections of the lungs (75 per cent., according to Whitman, and 70 per cent. of Janeway's collection of cases of the type of Marie and v. Bamberger). The forms of lung disease most commonly met with in osteoarthropathy are tuberculosis, bronchiectasis and other suppurative affections of the lung and pleura, although hydatids and malignant growths have been observed in a few cases.

The affection consists essentially in an ossifying osteoperiostitis limited usually to the distal extremities of the long bones (ulna, radius, tibia and fibula); less frequently of the carpal, tarsal and phalangeal bones. In severe cases the whole skeleton is affected, with kyphosis, usually in the lower part of the spinal column. The changes in the distal phalanges themselves are extremely slight, and are often not demonstrable even by the most careful radioscopy. In other cases a distinct osteophytic growth occurs, occasionally large cauliflower masses being observed at the ends of the phalanges. But the bony changes are not the only cause of the deformities observed during life; a large part of the alterations are due to thickening and swelling of the soft parts. The peculiar shape of the fingers, the characteristic clubbing, depends almost entirely upon hypertrophy of the soft parts. Radiography has taught us this. The same may be said of the swellings about the joints, which give the impression that the joint is affected, and to which the name arthropathy is due; much of this, doubtless, depends upon the enlargement of the bone ends, but much, also, upon hypertrophy of the soft parts. According to Freytag (Sternberg), the microscopic examination of the soft parts has thus far offered nothing of note.

F. R. Walters divides the affection into three groups: I. Typical osteoarthropathy, with peculiar form of clubbed fingers and changes in the long bones. II. Cases in which only the peculiar form of clubbed fingers exist. III. A mixed group, including all cases in which the enlargement of the extremities appears to have been primary, as well as others which are of uncertain nature. As a fourth group we would add the ordinary clubbed fingers of chronic heart and lung disease, especially congenital heart disease, as the latter are certainly in close clinical and etiological relationship with osteoarthropathy. Sternberg regards the ordinary clubbed fingers as the lightest grade of the affection. Walters,

also, considers the two conditions as closely allied, and remarks that the majority of cases have been associated with conditions that interfere with the aeration of the blood; again, v. Bamberger's cases show that valvular heart disease may cause changes in the bones as well as clubbing of the fingers. · The clubbing of the fingers in osteoarthropathy, however, is quite characteristic, and differs materially from that of chronic heart and lung disease (the so-called Hippocratic fingers). The nail is claw-like

PLATES I. AND II. PHOTOGRAPH AND RADIOGRAPH OF HANDS IN A CASE OF CONGENITAL HEART DISEASE, PATIENT'S AGE SEVEN YEARS.

and curved in both its lateral and longitudinal diameters; it strikingly resembles the beak of a parrot, and, from hyperextension and enlargement of the terminal phalanx, it assumes the shape of a drum stick; hence the name "trommelschlägel finger." "The enlargement," observes Walters, "is mainly antero-posterior and dorsal, whereas in ordinary clubbed fingers it is lateral and palmar. Intermediate forms, however, occur." The nail, too, in osteoarthropathy, is rose red in color, and is often striated and brittle, so that it splits readily, while in the Hippocratic finger the nail is apt to be blue

and cyanosed. The difference between the two forms will be readily appreciated by comparing plates I. and II., from a case of congenital heart disease, with III. and IV., representing our own case of osteoarthropathy limited to the finger ends. The deformity of the fingers usually occurs uniformly in all fingers and toes (Sternberg), although, as a rule, less marked in the latter. This form (that limited to the finger ends) is apt espe-

PLATE III. PHOTOGRAPH OF HANDS, CASE OF OSTEO-ARTHROPATHY, PATIENT'S AGE EIGHT YEARS.

cially to occur with chronic suppurative lung diseases, and is more common in children, but has been observed in adults. In empyema the Hippocratic finger was considered by the older writers of diagnostic importance (Eustace Smith). They appear quickly in this affection, and disappear, or diminish, as the original diseases improve. Of such character were the cases of Gillet, Moizard and Moussons; all in children. According to Wunderlich, the clubbed fingers are seldom painful, as is usually the case with the swellings of the long bones.

While Freytag and Whitman regard the ordinary clubbed

fingers as a distinct affection (and the peculiar shape of the osteoarthropathic finger would seem to support this view), we are inclined to believe with Janeway that it is "wise to consider the conditions as different stages of the same process until a case is found with the bone lesions and no clubbed fingers" (no case has as yet been observed without the drumstick finger), "or until a certain and different etiology is proven for the two manifestations."

As to the etiology and pathogenesis of osteoarthropathy, the

PLATE IV. RADIOGRAPH OF HANDS SHOWING CHANGES IN THE TERMINAL PHALANGES IN CASE OF PULMONARY OSTEOARTHROPATHY, SAME PATIENT AS PLATE III.

views of v. Bamberger and Marie that the changes are due to the absorption of poisons from the seat of primary disease, whether that be pulmonary, or a general infection or chronic intoxication, seems, in the main, to be the most probable; although it will not apply to congenital heart disease, for which, however, venous congestion may possibly account. Janeway believes that this factor has not been sufficiently accentuated, both as a cause of

ordinary clubbed fingers, and as an accessory in the cases of pulmonary or other origin. Thorburn attributed the condition to a diffuse tuberculosis; and a small group were thought by Teleky to have a nervous source, a view which, Janeway says, has no substantiation.

Osteoarthropathy is exceptionally rare in children, *i.e.,* the type of Marie and v. Bamberger. Because he found no cases in a children's hospital, Lefebvre believed it was always an adult disease (Whitman). The most typical case of the type of Marie and v. Bamberger that we are cognizant of is that of Whitman: a girl, who developed Pott's Disease at the age of two; at five there was pertussis, followed by persistent cough and expectoration; at five and one-half years, enlargement of the finger ends was first noticed; this was so marked at the age of six and one-half as to attract universal notice; at eight years, thickening of the lower arms and swelling of wrist joints, characteristic clubbing, moderate kyphosis and rigidity of spine, and signs of tuberculous disease of the lungs. Field's case at seventeen months is regarded by Sternberg and Janeway as osteoarthropathy, by Arnold as acromegaly. Davis' boy of four and one-half years, who developed characteristic changes in association with empyema, one year after pneumonia, seems to be a genuine case. v. Bamberger reported a case at seven years, with pulmonary stenosis, congenital cyanosis, and tubercular lung deposits, and Thorburn refers to a post-mortem in a boy (age not stated), with thickening of the tibiæ, fibulæ, radii and ulnæ, with ordinary clubbed fingers, mitral stenosis and no lung disease. Gillet saw a typical case in a lad of thirteen, who had had repeated bronchitis since the age of four. We thus have only 5, or at most 6, typical cases of Marie's type in children.

Of cases in which the characteristic (parrot-beaked) changes were limited to the finger ends there have been reported in children: one by Moussons, a girl of fourteen, with empyema; 1 by Gillet, girl of seven and one-quarter, also with empyema; Moizard's 2 cases, a boy of six, and a girl of five, the first with purulent pleurisy and vomica, the second with empyema; Marfan's 3 cases, age not stated, one with empyema, one with bronchial dilatation, and one with cystitis and right bronchial dilatation; finally, Rotch and Dunn's case in a boy of three years and seven months. These observers, however, are in error when they class their case as one of osteoarthropathy, because "it cor-

responds to the description given of pulmonary osteoarthropathy; that is, enlargement of the entire distal phalanges, involving primary the bone itself"; since it has been abundantly shown that, even in typical cases, the distal phalanx itself is rarely affected, the deformity being almost entirely due to hypertrophy of the soft parts. When it does exist, however, we believe it places the case in the category of v. Bamberger and Marie, even should the characteristic clubbing not be present; a combination which, as yet, has not been observed.

If we class the ordinary clubbed fingers of chronic lung and heart disease as the lightest grade of osteoarthropathy, the number of cases occurring in early life will be greatly enlarged, as congenital heart disease alone would materially swell the list. Gillet believes that in childhood the affection is limited to the terminal phalanges, and displays little tendency to invade the larger bones; but Whitman's case apparently proves that, although this may usually be the rule, the Hippocratic fingers may occasionally develop into typical osteoarthropathy; in this case there was a period of five years between the first appearance of the clubbed fingers and the signs of implication of the long bones.

While our own patient was under observation we met with a boy of eight years, who had Pott's Disease and chronic bronchitis, with clubbing of the fingers slightly suggestive of the form peculiar to osteoarthropathy, but in whom radioscopy revealed entirely normal phalanges. It is our intention to observe whether the case which we now present, as typical of osteoarthropathy limited to the distal phalanges, will in time develop osteoperiostitis of the long bones and other features of the type of Marie and v. Bamberger.

F. W., a girl, was first seen at the age of eight years. She had been deformed since birth, but had become worse during the few months prior to admission. The family history was negative. There was marked kyphosis, embracing the first seven dorsal vertebræ, with a compensating anterior curve from the latter to the sacrum. The thorax was quite prominent, the ribs distinct and the sternum pushed forward. There was a marked rosary, distended abdomen, and emaciated arms and legs. The facial expression was that of an old woman. At this time, February 5, 1903, the condition of the fingers was not noted, although clubbing was probably present in some degree. The urine was normal; the temperature 99° to 100°-101° F.; there was hectic, cough and signs

of general bronchitis. After a long period in bed the child was permitted to go about with a jacket. The writer saw the patient first in February, 1904, about one year after admission to hospital. The clubbing at that time was marked and quite characteristic, the nails curved like a parrot's beak, elevated, of a rosy red hue and brittle. The fingers were long, and from hyperextension and enlargement of the terminal phalanges, resembled closely a drumstick. The following measurements were made:—

MIDDLE FINGER, LEFT HAND.	CM.
Circumference first phalanx	4.1
" middle "	3.6
" last "	4.5
Length of nail	1.2
Breadth of nail	1.5

THUMB.	CM.
Circumference first phalanx	4.4
" last "	5.1
Breadth of nail	1.8
Length of nail	1.6

The toes presented conditions similar to that of the fingers, but very much less marked. There was absolutely no thickening of the radii, ulnæ, tibiæ or fibulæ, both objectively and by means of radiographic pictures; the joints were entirely unaffected, nor had there ever been any pain in the latter or in the deformed fingers. The face was broad, and the head large and square, the molar bones prominent and the teeth carious. The child was quite anemic, the skin being of a peculiar muddy hue, but the blood count showed only a moderate secondary anemia: R. B. C., 4,250,000; W. B. C., 17,000; Hg. 52 per cent. Differential count: polymorphonuclear cells, 82.8 per cent.; large lymphocytes, 12.4 per cent.; small lymphocytes, 3.4 per cent.; eosinophiles, 1.4 per cent. The patient had had a cough for a long time; it varied in intensity, but was never entirely absent. Fever was also more or less constantly present, usually above normal, but occasionally falling below the latter point; as a rule, the diurnal range was between 99° and 100° to 101° F. The pulse was 100; respiration 28. The urine contained neither sugar nor albumin; no casts; specific gravity, 1,020; acid. An examination of the chest revealed the following: Heart dullness

extended from third rib to fifth interspace, nipple line (apex); right border at middle of sternum; sounds heard with great distinctness all over left chest; no murmurs. Lung dull at right supraspinous fossa, becoming quite clear as base is approached, and quite clear at base itself; at left supraspinous fossa slightly impaired resonance, remainder of right side, posteriorly, clear; at right supraspinous fossa feeble and distant bronchial breathing, similar to that heard over an effusion or compressed lung, increased voice and pectoriloquy, and a few crackling râles, elsewhere right lung shows harsh breathing and subcrepitant râles; at left supraspinous fossa harsh breathing only, no râles. Anteriorly, due to second rib on right side, with harsh breathing and no râles; at the left apex, in front, tympanitic note and bronchovesicular breathing, no râles. The liver extended a finger's breadth below the costal border; the spleen was enlarged and palpable. The pulmonary condition continued during the two months the child was under observation. How much the bronchial breathing heard at the right apex was due to compression from the greatly deformed chest, we are not prepared to say; but its character and persistence makes it quite possible that it had its origin in an old and healed cavity, or, possibly, to interstitial changes, the patient, at any rate, had a chronic bronchial catarrh, which may or may not have been tuberculous. No sputum could be obtained for examination. Equally uncertain is it to determine how much of the bony changes were the result of the pulmonary affection, on the one hand, and the chest deformity on the other; probably both contributed their share. The radiographs of the thorax were so unsatisfactory that the condition in the lung could not be determined by this method. Those of the hands, however, showed distinct periostitis of the terminal phalanges of the hands and feet, especially of the former, where definite osteophytic growths, somewhat cauliflower in shape, can clearly be seen, thus placing the case in the category of those true osteoarthropathies, in which the deformities are limited to the distal phalanges, in contradistinction to the closely allied, but not exactly similar, clubbed finger of chronic heart and lung affections.

NOTE.—After the proof of this paper had gone to the printer the patient died. The examination was limited to the thoracic and

abdominal cavities, permission to dissect the affected finger-ends not being obtained. There was marked interstitial pneumonia and purulent bronchitis, lesions sufficient to account for the phalangeal changes observed during life.

Abstract of autopsy by W. E. Robertson, M.D.:

Pleural adhesions both sides, especially about the apices, and between the bases and the diaphragm, interlobar adhesions also. Except at the apices, none of these were especially dense, nor was there thickening of the pleura.

Lungs.—Apices markedly fibrotic, and not compressible, no crepitation, interstitial pneumonia. This latter was certainly not pleurogenous in origin, but probably peribronchial, though sections have not yet been made. Signs of purulent bronchitis. No evident tubercules either in the lungs or peribronchial lymph nodes, but the latter were enlarged and pale. There were enlarged glands in the posterior mediastinum on the left side to which the apex of the lung was densely adherent. About this considerable fibrous tissue existed, possibly sufficient to act as an obstructive factor during life.

Heart.—No gross lesion except slight dilation. Mesenteric adenopathy. Liver and spleen enlarged. Other abdominal organs normal.

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THE WORK OF THE BABIES' WARDS OF THE NEW YORK POST-GRADUATE HOSPITAL FOR CONVALESCENT CHILDREN.

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The collection of infants and young children in a hospital presents many problems beyond the mere medical treatment of the cases. As far as lasting good to the children is concerned, the purely medical function may be the least important feature. The reason of this is twofold—first, the effect of the hospital itself upon the child; and, second, the essentially faulty life conditions that send the child to the hospital. Unless these two factors are attentively considered, the results of care and treatment will often prove unsatisfactory and ephemeral. Work in any one line of charity, if properly conceived, almost invariably opens up new and converging lines of effort that are necessary to employ if the work is to be made permanent. To treat a child successfully for pneumonia in a hospital, and then send it to damp and unhygienic rooms in a tenement house, is but to invite another attack of the original disease; hence, the housing problem looms up for consideration. Again, after relieving an infant of gastroenteric irritation in hospital or dispensary, the work is only half done unless steps are taken to procure pure, fresh milk for future feeding, so that the food problem must be considered. As the hospital is not a good place for recuperation, when the patient is discharged the work will often fail in lasting results unless the child can be thoroughly invigorated, and here convalescent homes in the country at once claim attention.

Hospital statistics, as such, are of little value unless they take into consideration the ultimate condition of the patient. Until hospital managers are able or willing to extend their work along

these lines of permanency, or have agencies that will do so, their work fails in much of its possibilities. No place more than a hospital gives the chance to trace needs and opportunities to their ultimate sources. It should not alone be a place for treating the sick, but a social laboratory, where future prevention should be constantly studied in connection with the cases under observation.

In considering the lasting good of the child, the hospital itself merits the first consideration. For infants and very young children it should simply serve as a temporary place of refuge, where all hygienic, medicinal and surgical appliances should be of the latest and best types. After the acute illness has subsided, the case should be immediately removed, as, otherwise, there will be constant relapses and reinfections. The latter will occasionally occur even with the best nursing and under the finest hygienic surroundings in the hospital. No hospital should admit infants or very young children to its wards unless all the appliances are of the very best as regards hygiene, air space, ventilation and skilful nursing. The nurses must likewise be sufficient in number, one to 5 or 6 cases, and specially trained in this line of work. Even where all these conditions are fulfilled, the greatest care and watchfulness will be required and the infants must be discharged at just the right time, when the acute symptoms have subsided and before a general cachexia or atrophy has had time to develop. In order then to ensure a good convalescence, the infant must be kept in the hospital for only a short time, it must be carefully guarded from auto- and hetero-infection while there, and, finally, sent out to recuperate under favorable conditions. When detained too long, certain phenomena invariably develop. Those especially to be noted are a slow but progressive loss of weight not entirely dependent on the original disease, dryness of the skin, hydremia, hypostatic pneumonias of a peculiarly insidious character and great susceptibility of the mucous membranes. The latter are specially shown by bowel irritation, and vaginitis in female infants.

At the Babies' Wards of the Post-Graduate Hospital, forming a separate division of the hospital, the greatest care is exercised to avoid the dangers of prolonged hospital environment. Constant vigilance is employed in preventing infection from entering or spreading. On admission the clothing is removed and re-

tained in the examining room and the child given an antiseptic bath before being taken to the wards. All female children have a smear taken from the vagina, which is immediately examined in order to ensure freedom from the latent infection which is so frequently found in these parts. No child is admitted from a house where any contagious disease exists, slips furnished by the Board of Health being constantly consulted. When a mother visits her child she is furnished a gown before being admitted to the wards. All stools and napkins are promptly disinfected. No mattresses are employed, the bedding consisting of woven wire springs that can be washed, covered by blankets. When the child is discharged, all the bedding and clothing are placed in a large sterilizer and subjected to live steam.

The wards are large, sunny, well-ventilated, and with all the best and most modern hygienic devices. The infants are daily removed to a sun parlor at the top of the building in order to get a complete change of air, and, when the weather permits, to a roof garden. The plant here in existence and the plan of treating the cases now serve as a model for many similar institutions throughout the country.

In order to make permanent the benefits of the hospital, the infants are kept only during the period of acute illness, and then discharged, but still kept under observation outside. This system started with the plan of simply having a visitor, but has eventuated in methods of fresh-air treatment that are aimed to ensure a thorough convalescence, if such a thing is possible. In 1890, a voluntary committee of three ladies, at the request of the writer, undertook to visit and report on the cases discharged from the hospital. As the work grew in importance it became evident that a more systematic effort was required, and, accordingly, a special committee was formed from the Ladies' Auxiliary Committee in 1894. A paid visitor, who could work at all seasons, was appointed, and this plan has been in operation ever since. During the past five years a female physician has done this work. A combination of medical knowledge with tact and observation here give the best results. A blank that is of proper size to be attached to the regular hospital history is filled out by this visitor, so that the attending physician can at once be posted as to the life conditions of the child, and hence be governed as to the time of discharge from the hospital and what steps may be necessary to

take in order to improve faulty conditions. The following items are placed upon this chart:

Name.
Address.
Age.
Number of rooms, No. light, No. dark, No. on air shaft.
Sanitary condition of house.
Sanitary condition of street—Fault landlord or city.
Physical condition of parents.
Intelligent care of children at home.
Condition of child second week after return to home.

A full history of the child's parentage and personal condition having been already taken by the examining physician on admittance, a pretty complete record is in the hands of the attending physician for his guidance in managing the case.

The visitor has placed in her hands a certain sum of money to purchase clean, fresh milk, when necessary, and to relieve any pressing needs that may interfere with the child's convalescence. She also sees that the food is properly prepared, medicines given, and the hygienic surroundings improved as much as possible. Rules of instruction in feeding and hygiene for mothers, translated into German and Italian, have proved of great value. The work of the visitor has been in many other ways of far-reaching utility. Landlords have been brought to terms in putting premises in a sanitary condition rather than be reported to the Board of Health; fewer children are deserted than formerly, as a special effort is made to make the mothers realize the crime of deserting their children; all existing charities are made use of in as far as they apply to the case in hand, such as the free ice in summer, depots for milk distribution and all the fresh-air agencies. The mothers have explained the great advantage of allowing the children to have a change, even although it be only for a day, but efforts are made for a longer outing. It is believed that this work has been of the greatest utility to our convalescent children during the ten years that it has been systematically employed. Many facts of great social interest have also been brought out. Some of the records have been carefully studied and tabulated. Two tables have been thus prepared, covering different periods.

TABLE NO. 1.
HOSPITAL STATISTICS.

BEING A RECORD OF 1,000 CASES, EXTENDING FROM MARCH, 1900, TO MARCH, 1902.

CONDITION OF STREET.		CONDITION OF HOUSE.		CONDITION OF ROOMS.	
Good	464	New and clean	200	Good	405
Fair	157	Old and clean	245	Bad	161
Bad	153	New and dirty	19	Fair	205
Not indicated	226	Old and dirty	197	Not indicated	229
	1,000	Good	89		1,000
		Bad	37		
		Not indicated	213		
			1,000		

TOTAL NUMBER OF ROOMS.		NUMBER OF LIGHT ROOMS.		NUMBER OF DARK ROOMS.	
1 Room	58	1 Room	384	0 Room	93
2 Rooms	165	2 Rooms	169	1 "	208
3 "	327	3 "	58	2 Rooms	203
4 "	168	4 "	46	3 "	46
5 "	59	5 "	7	4 "	3
6 "	12	6 "	3	5 "	0
Not indicated	211	Not indicated	331	Not indicated	447
	1,000		1,000		1,000

NUMBER OF ROOMS ON AIR-SHAFT.		NUMBER OF BOARDERS OR LODGERS.		PARENTS' DRINK HABITS.	
0 Room	207	0 Lodger	386	Good	338
1 "	49	1 "	69	Bad	31
2 Rooms	104	2 Lodgers	39	1 drinks	36
3 "	37	3 "	12	Not indicated	595
4 "	2	4 "	3		1,000
Not indicated	541	5 "	5		
	1,000	Not indicated	486		
			1,000		

PARENTS' AVERAGE EARNINGS.		PARENTS' NATIVITY.		PARENTS' RELIGION.	
$1–5	173	American	231	Catholic	365
5–10	379	German	143	Jewish	242
10–15	152	Irish	94	Protestant	52
15–20	12	Other Nations	532	Mixed	236
Not indicated	284		1,000	Not indicated	105
	1,000				1,000

MARRIED, WIDOWED, OR DESERTED OR SINGLE.		NUMBER OF OTHER CHILDREN.	
Married	724	0 Others	202
Widowed	79	1 "	213
Deserted	90	2 "	202
Single	7	3 "	95
Not indicated	100	4 "	78
	1,000	5 "	28
		8 or more	54
		Not indicated	128
			1,000

TABLE NO. 2.

HOSPITAL STATISTICS.

BEING A RECORD OF 700 CASES, EXTENDING OVER A PERIOD OF ONE YEAR, FROM MARCH, 1903, TO MARCH, 1904.

SANITARY CONDITION OF STREET.		SANITARY CONDITION OF HOUSE.		NUMBER OF ROOMS.	
Indicated in only 146 Report Cards.				1 Room	25
				2 Rooms	64
Good	33	Good	125	3 "	115
Fair	21	Fair	88	4 "	74
Bad	18	Bad	96	5 or more	29
Not indicated	74	Not indicated	391	Not indicated	393
	146		700		700

NUMBER OF LIGHT ROOMS.		NUMBER OF DARK ROOMS.		NUMBER OF ROOMS ON SHAFT.	
1 Light room	137	1 Dark room	72	1 Room	8
2 " rooms	64	2 " rooms	91	2 Rooms	38
3 or more	93	3 " "	11	3 or more	11
Not indicated	406	Not indicated	526	Not indicated	643
	700		700		700

NUMBER OF LODGERS.		AGE OF CHILD.		NUMBER OF OTHER CHILDREN.	
1 Lodger	17	6 Months or less	229	0 Others	138
2 Lodgers	17	1 Year	118	1 "	171
3 "	9	2 Years	156	2 "	123
Not indicated	657	3 "	73	3 "	76
	700	4 "	64	4 "	54
		5 and over	60	5 or more	64
			700	Not indicated	74
					700

HOME CARE OF CHILDREN.		PHYSICAL CONDITION OF PARENTS.		AVERAGE EARNINGS OF PARENTS.	
Good	105	Good	486	$1–5	84
Fair	97	Fair	11	10	220
Bad	105	Bad	103	15	112
Not indicated	395	Mixed	79	20 or over	48
	700		700	Not indicated	236
					700

NATIVITY OF PARENTS.		RELIGION OF PARENTS.		MARRIED, WIDOWED, DESERTED OR SINGLE.	
American	168	Catholic	326	Married	533
Irish	48	Jew	152	Widowed	53
German	54	Protestant	144	Deserted	59
Other Nations	307	Mixed	46	Single	13
Mixed	123	Not indicated	32	Not indicated	38
	700		700		700

While it is impossible to get complete data in all cases, it is believed that these tables will give some idea of the work actually done and the good accomplished.

The efforts of the hospital to aid its convalescent children do not end with the work of the professional visitor and the help she can muster. Two other special agencies are constantly employed, which, although not under the same management as the hospital, have grown out of its work and are closely affiliated with it.

A fresh-air home at Sea Cliff, L. I., specially built and equipped for convalescent children, is open from May to November, and takes all needed cases for intervals of from two weeks to several months. It is situated on a high promontory, sufficiently wooded, with a porous soil, overlooking Long Island Sound. Everything that can conduce to healthy, outdoor living is here supplied. A resident physician and nurses, specially trained in the Post-Graduate Hospital, keep careful oversight of the cases, and no child is discharged until it is believed that the improvement will be permanent.

Several years ago the author resolved to try the plan of boarding out in the country bottle-fed infants who were not doing well in institutions or in their homes. At his request several ladies started the nucleus of such a work, which has since grown.

The Speedwell Society, located at Morristown, N. J., a very healthy district, has resulted and is in operation during the whole of the year. The plan of this Society is to board out infants and very young children in country homes in the vicinity. The success of this method of placing bottle-fed infants in private homes where they can have individual attention has been thoroughly demonstrated during the three years of the operation of this work. This is true even if the woman taking the baby into her own home is fairly ignorant. The mothering she gives the infant often makes up for her lack of accurate knowledge, and this is especially true in marasmus cases. However, a paid physician and two trained nurses keep these cases under constant observation. It has been found that bottle-fed infants and cases of general malnutrition do better under this plan of treatment than in the best-managed institutions. Many of our youngest cases have thus been completely restored to health and strength after the acute illness has been relieved in the hospital.

PRIMARY ADENOSARCOMA OF THE LIVER IN A CHILD OF NINE MONTHS.

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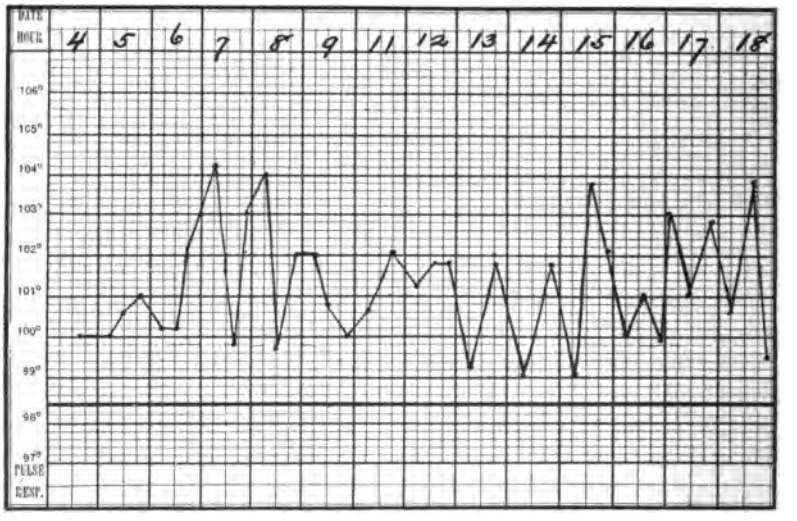
The patient was a male child admitted to the Babies' Hospital April 4, 1904, on account of an abdominal tumor.

The parents were both living and healthy, also one other child; there was a history of tuberculosis in the father's family, but nothing else which bore even remotely upon the case could be ascertained. The patient was born at term after an easy labor, had been breast-fed and was reported always to have been a pale child but well nourished and had gained steadily in weight. The child did well up to the age of seven months when, on account of some digestive disturbance, chiefly vomiting, bottle-feeding was substituted for part of the nursings. The vomiting had recurred at varying intervals until the patient was admitted. The stools were green and undigested and the child lost weight, though not rapidly. The only symptoms noticed had been this disturbance of digestion until the week before admission, when a tumor of the abdomen was discovered on account of which the child was sent to the hospital.

The examination on admission showed a well developed but pale and rather poorly nourished child; weight 15¼ pounds; four teeth; nothing abnormal in heart or lungs. The abdomen was moderately distended, being distinctly more prominent on the right side than on the left. Here was felt a large tumor forming a hard mass extending, at the ensiform cartilage, about two inches to the left of the median line; at the umbilicus, a little to the left of the median line. Its lower border extended in a curved direction from just below the umbilicus to the crest of the ilium. Its surface was smooth and semi-elastic. Continuous flatness on percussion extended from the sixth rib to the lower border of the tumor. A mass was felt in the left side of the abdomen corresponding to the spleen. Neither by palpation nor percussion could the liver be made out as separate from the tumor. The skin

over the abdomen was normal except for a slight distension of the superficial veins. No other masses could be felt in the abdomen or in the pelvis; genitals normal. There was slight edema of both feet and legs; no paralysis. There were no nodular enlargements except slight ones in the cervical region. The examination of the blood showed hemoglobin, 55 per cent.; leukocytes, 17,000; fresh blood, normal.

An exploratory needle was passed into tumor about its middle and 2 cc. of bloody fluid withdrawn. The microscopical ex-



TEMPERATURE CHART IN CASE OF ADENOSARCOMA OF THE LIVER. THE CHART SHOWS THE COURSE OF THE TEMPERATURE AFTER THE OPERATION.

amination showed, red blood cells, fatty liver cells, fat droplets and small round mononuclear cells. Culture sterile.

After the child had been under observation for three days, as it was growing steadily worse, exploratory incision was decided upon. Up to this time slight fever had been present 100°-101°F., and occasionally vomiting, but there was no jaundice and no absence of bile from the stools was noticed. The child looked very sick; took food very badly.

The operation was performed by Dr. A. L. Fisk under cocain anesthesia. A transverse incision was made about two inches be-

low the costal margin over the most prominent part of the tumor. The tumor was found to be, as had been suspected, an enlarged liver, surface smooth and glistening. The liver was aspirated in various directions, but no pus found; but at one point, which seemed softer than the rest, an opening was made and a drainage tube inserted into the substance of the organ. This passed into a cavity apparently made up of broken down liver tissue as from it a grumous material was discharged, but no pus.

Considerable shock followed, but on the whole child bore it very well. After the operation the child lived thirteen days with a continuous but irregular temperature (see chart), steadily increasing prostration and gradual though not rapid loss in weight. Five days after the operation the leukocytes were 13,000. There were no local symptoms of importance, the wound was dressed daily and did well. The tumor did not change essentially in appearance. There was occasionally vomiting but no diarrhea and little abdominal distension. The liver seemed slightly smaller during the last few days. Death occurred from exhaustion.

PATHOLOGICAL REPORT BY MARTHA WOLLSTEIN, M.D.

Autopsy, twelve hours after death. Body, well nourished; edema of feet and hands; operation wound 4 cm. long to the right and above the umbilicus. Wound normal.

Liver.—Measures 19 cm. long, 21 cm. wide, 10 cm. thick; weight, 735 grams; of a light brown color, anterior surface mottled, with lighter and darker areas. There was a rounded, bulging and apparently fluctuating mass measuring 9 x 7 cm., which projected somewhat above the level of the rest of the organ. This extended nearly to the lower border of the liver. The lower border of the liver is sharp not rounded. The relation of the tumor to the rest of the liver is well shown in the section from which the photograph (see illustration) was taken. The operation wound, 2 cm. long, penetrates the softened prominent tumor mass at its centre. The anterior part of this protruding tumor fluctuates, but this is due to necrosis; the rest of the tumor, exposed by section of the liver, is a firm, nodular, yellowish-white color mottled with larger and smaller hemorrhagic areas and separated from the liver substance by thin capsule. The softened portions are limited to the anterior part. In the interior there are found only a few scattered softened nodules, some of which are black in color from hemorrhage. The liver substance outside

the growth is firm and moderately fatty. The posterior surface of the liver shows a mottling due to the presence of the tumor.

ADENOSARCOMA OF THE LIVER: SECTION SHOWING RELATION OF THE TUMOR.

Near its upper border are three small separated yellow nodules. The liver capsule is smooth, there is no peritonitis and apparently

no thickening of the interlobular connective tissue septa. Gall bladder 4 cm. long contains fluid bile; ducts are all pervious.

There was a slight recent pericarditis and a moderate bronchopneumonia. All the other viscera were essentially normal, except the kidneys which showed slight recent nephritis, no metastasis being present anywhere.

Cultures from the heart, liver, spleen and kidneys gave a pure growth of the streptococcus pyogenes. This was evidently a terminal infection.

Microscopical Examination.—In sections from the periphery of the tumor, the capsule is seen to be composed of a layer of cirrhotic liver substance in which newly-formed bile ducts are found in small numbers. The growth is made up of lobules of liver cells, arranged in atypical columns; the central vein is often at one side of the lobule, and again it cannot be found. Small interlobular blood capillaries are numerous, and many small hemorrhages have taken place in and between the lobules. The epithelial cells show many karyokinetic figures.

Sections from the softened hemorrhagic portion near operation wound showed two varieties of cells. The epithelial cells resembling those of the liver are arranged in the adenomatous lobules as in the rest of the tumor. In addition there are irregular, larger and smaller masses of round cells whose nuclei stain deeply and are comparatively large. These cells lie in a very delicate stroma of connective tissue fibrils. Small blood vessels are very numerous, and hemorrhages into the tissue have taken place. The softened areas seen in the gross specimen are due to large hemorrhages in this portion of the growth which is evidently a small round cell sarcoma. The sarcoma nodules lie between the adenomatous ones and in places they grow into the latter, *i.e.*, between the columns of epithelial cells are irregularly-sized sarcoma nodules. At the capsule the sarcoma cells infiltrate the connective tissue for some distance in contrast to the adenoma which is sharply limited.

The liver substance outside the tumor maintains its lobular structure perfectly. The liver cells contain smaller and larger fat droplets, especially at the periphery of the lobules. The connective tissue septa are not thickened—there is no general cirrhosis.

Remarks.—No exactly similar case can be discovered in medical literature. But two liver adenomata in children have been

found recorded: one aged twenty months in which Weichselbaum[1] made the microscopic examination, and the other in a boy of twelve years, described by Pye-Smith.[2]

The case here reported would come under the head of the *hepato adenoma proprium* of Witwicky,[3] the cells of the neoplasm originating from those of the liver, as distinct from the epithelium of the bile ducts, and being arranged in columns. Such growths do not become malignant and form no metastases.

Primary congenital sarcomata of the liver have been described by Pepper[4] in an infant eight weeks old; by Heaton[5] at the same age; by Parker[6] in a baby of three weeks, Pepper[7] at seven weeks, Gee[8] at five months and by De Ruyter.[9] All of these were round-celled sarcomata. Meisenbach's[10] case, four months old, was a myxosarcoma. Windrath[11] describes a spindle-cell sarcoma in a baby less than one year old. Bauman and Forbes' case[12], eleven months old, was a round and spindle-celled sarcoma. West's[13] case, eight months old, was a medullary sarcoma. Henoch[14] gives no microscopic details of the sarcoma he reports in a child of two and a half years. Roberts[15] reports a medullary sarcoma in a girl twelve years old. Several of these cases were accompanied by metastases in the suprarenal capsules and in the lung. In addition to these primary cases quite a large number of secondary sarcomata in the liver in children have been reported.

The combination of adenoma and sarcoma would seem to be unusual among the tumors of the liver occurring in infants. The presence of a distinct capsule around the whole neoplasm, and, the fact that the sarcoma is not found in all parts but only in the central zone, would seem to argue that the adenoma was the original process, to which the sarcoma was added at a somewhat later date.

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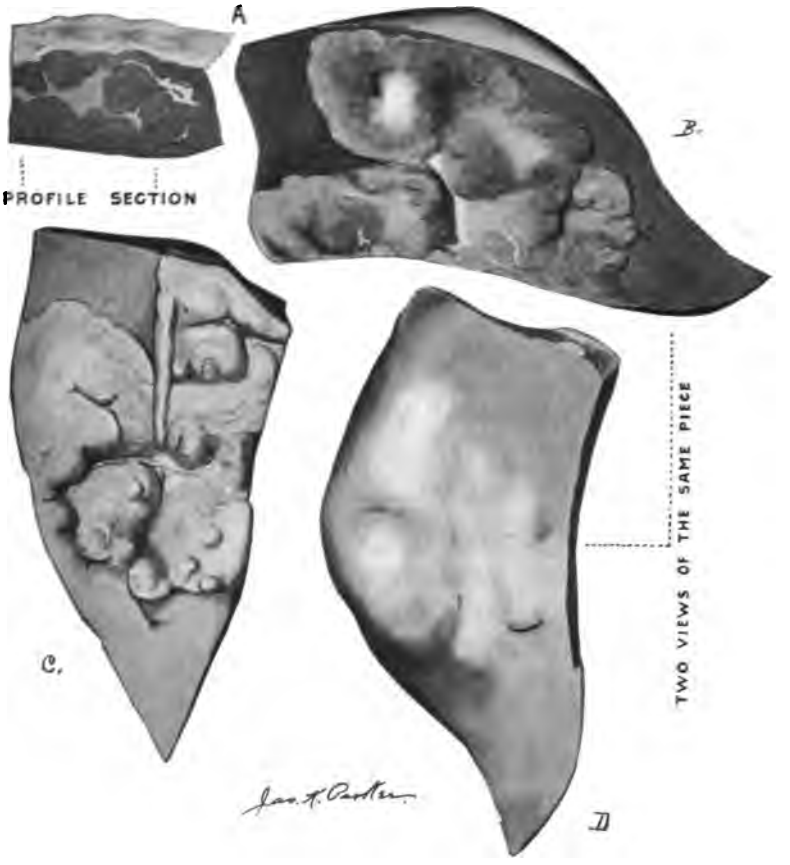
DISCUSSION.

DR. ABT.—In an autopsy on a female child, aged twenty-one months, who died of typhoid fever, complicated by multiple gangrene of the skin, an adenoma of the liver was found, which was unsuspected during life. In addition to the adenoma, the liver showed catarrhal cholangitis, which had no connection with the adenomatous condition. Upon examining the liver, it was observed that in the interior portion of the left lobe there was an area nearly the size of a lemon which stood out quite sharply from the surrounding tissue, and was irregularly circular in outline. The central portion of the growth was of a white color and seemed to be composed of fibrous structure. The periphery of the tumor was more yellowish or liver colored in appearance; it was raised and nodular. The larger nodes were half the size of a dime, the smaller ones the size of a pea. Microscopic sections, from the tumor, showed columns of liver cells which were not arranged in the form of acini. These columns of cells were separated from each other by trabeculæ of connective tissue. The connective tissue bands seemed continuous with Glisson's capsule. The individual cells were of very large size and many of them contained fat globules.

The question may arise whether this tumor should be considered an adenoma or simply a nodular hyperplasia. Ziegler believes that these tumors are true adenomata, although he thinks that there is no sharp line to be drawn between these and nodular hyperplasia on the one hand, and an adenocarcinoma on the other. Orth believes that a true hyperplasia of the liver parenchyma may occur. He has observed them in cases where a large portion of the liver has been destroyed by abscess, syphilis or echinococcus. Orth has observed hyperplasia resembling an adenoma in granular atrophy, and in a case of thrombosis of the hepatic vein. The same condition may occur in cirrhosis. This

hyperplasia may present a nodular appearance, or it may be single or multiple, the nodes varying from a pea to a cherry in size. Orth emphasizes the point that these hyperplasias, or adenomata,



ADENOMA OF THE LIVER, DR. ABT'S CASE.

FIGURE D.—THE APPEARANCE OF THE TUMOR AS SEEN ON THE LIVER CAPSULE.
FIGURE B.—CUT SECTION SHOWING THE NODULAR ARRANGEMENT WITH FIBROUS STRUCTURE BETWEEN THE NODULES.
FIGURE C.—CUT SECTION SHOWING LARGE AND SMALL NODULES OF THE TUMOR.

may eventually become carcinomatous. It may be mentioned in this connection that in the case which we report, there was no evidence of malignancy. The tumor was circumscribed, the cells did not invade the connective tissue, and there were no metastatic growths.

The occurrence of adenomata in the livers of children must be regarded as rare. Musser has reported 2 cases, one in a boy of twelve years, and another in an infant twenty months old. Birsch-Hirschfeld in 400 sections, in newborn children, observed adenomatous nodules in 2 cases. These adenomata could be recognized because they stood out prominently, also on account of their pale color; they were sharply circumscribed and a connective tissue capsule occurred on the periphery of the tumor. Microscopically, the tumors were made up of liver cells, the cells themselves showing granular changes and containing several nuclei. The significance of these adenomata permits of considerable speculation. If we consider them in the light of Cohnheim's hypothesis, which holds that retained embryonal tissue may cause neoplastic growth, then we may understand how the tumors may gradually change from simple to malignant adenomata. Observation has shown that these nodules take on a progressive character, *i.e.,* they grow constantly larger in size. and the exact moment when benignancy ends and malignancy begins, is impossible to determine. The case which was examined by Birsch-Hirschfeld is of interest in this connection. A boy twelve years old, presented a tumor of the liver, which had persisted for more than a year. Previously he had been perfectly well. The first sign of illness was indicated by a loss of appetite with vomiting and diarrhea. This condition was followed by ascites; after this, he lost rapidly in nutrition, and death occurred as a result of extreme marasmus. During the whole period of the illness, there was but slight icterus. A microscopic examination of the tumor showed that in certain areas the liver cells retained their radial type and presented a normal appearance, while in other areas, the cells were arranged irregularly, the interstitial connective tissue was increased and was very vascular. The cells invaded this connective tissue, which indicated in itself the malignant nature of the tumor.

A CASE OF ABDOMINOTHORACOPAGUS.

A Communication to the Society.

BY HERMAN B. SHEFFIELD, M.D.,

New York.

R. B., white, twenty-seven years old, was taken with labor pains on the 5th of March, 1904, at 7 A.M. I was sent for at 6 P.M., and found the membranes intact, the os fully dilated, R. O. P. position, and the labor pains very strong. The second examination two hours later revealed exactly the same condition as on the first examination. I ruptured the membranes and an enormous quantity of liquor amnii escaped, but without any distinct descent of the fetal head. Forceps were resorted to at 11 P.M., with the result that the presenting parts were delivered as far as the shoulders, and repeated attempts to deliver the remainder of the body utterly failed. Realizing the extraordinary condition of the case I decided to discontinue the anesthesia and to give Nature another opportunity. About one hour later the feet of the second baby were found to have descended near the nates of the first one. I then flexed the delivered parts of the first baby against the pubic arch and abdomen of the mother and by simultaneously exerting powerful traction, succeeded in delivering the first baby. In the meantime the second child assumed a more or less transverse position. I passed two fingers into the uterus, grasped the feet of the second baby and managed to deliver it without involving the slightest injury to the mother. The placenta (one) which was moderately large in size was delivered by Credé's method. The babies were fully developed and weighed 11½ pounds. There was but one umbilical cord. Part of the intestines prolapsed through the umbilical ring. The fetal heart sounds were distinctly audible until about a half an hour before complete delivery.

The accompanying illustrations give a complete picture of the mode of union, rendering a minute description of the monstrosity superfluous. Mention will be made only of the fact that a thin

layer of cartilage—representing the sternum—intervened between the opposing ribs. (See Figs. I. and II.)

The history of the mother lacks any important data to elucidate the etiology. The mother gave birth to a normal child two years

FIG. I.—PHOTOGRAPH OF CASE OF ABDOMINOTHORACOPAGUS.

ago. There were several twin births in the family and one of her sisters was delivered of living triplets three years ago. When the mother was four months pregnant she attempted to induce abortion by means of drugs and by lifting and dragging heavy pieces

of furniture. Her failure in the attempt was followed by a very exciting attack of hysteria of thirty-six hours' duration. From the fifth month on pregnancy proceeded without any unusual manifestations. The mother often felt active motion on both sides of the abdomen while the central portion always seemed to remain

FIG. II.—RADIOGRAPH OF CASE OF ABDOMINOTHORACOPAGUS.

stationary; she felt as though "two bodies were trying to tear themselves apart."

She made a perfect and rapid recovery.

I am indebted to Dr. H. Grad for the splendid radiograph and to Dr. G. Brown for his skilful assistance.